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COMMON AFFECTIONS
OF THE LIVER

W. HALL WHITE

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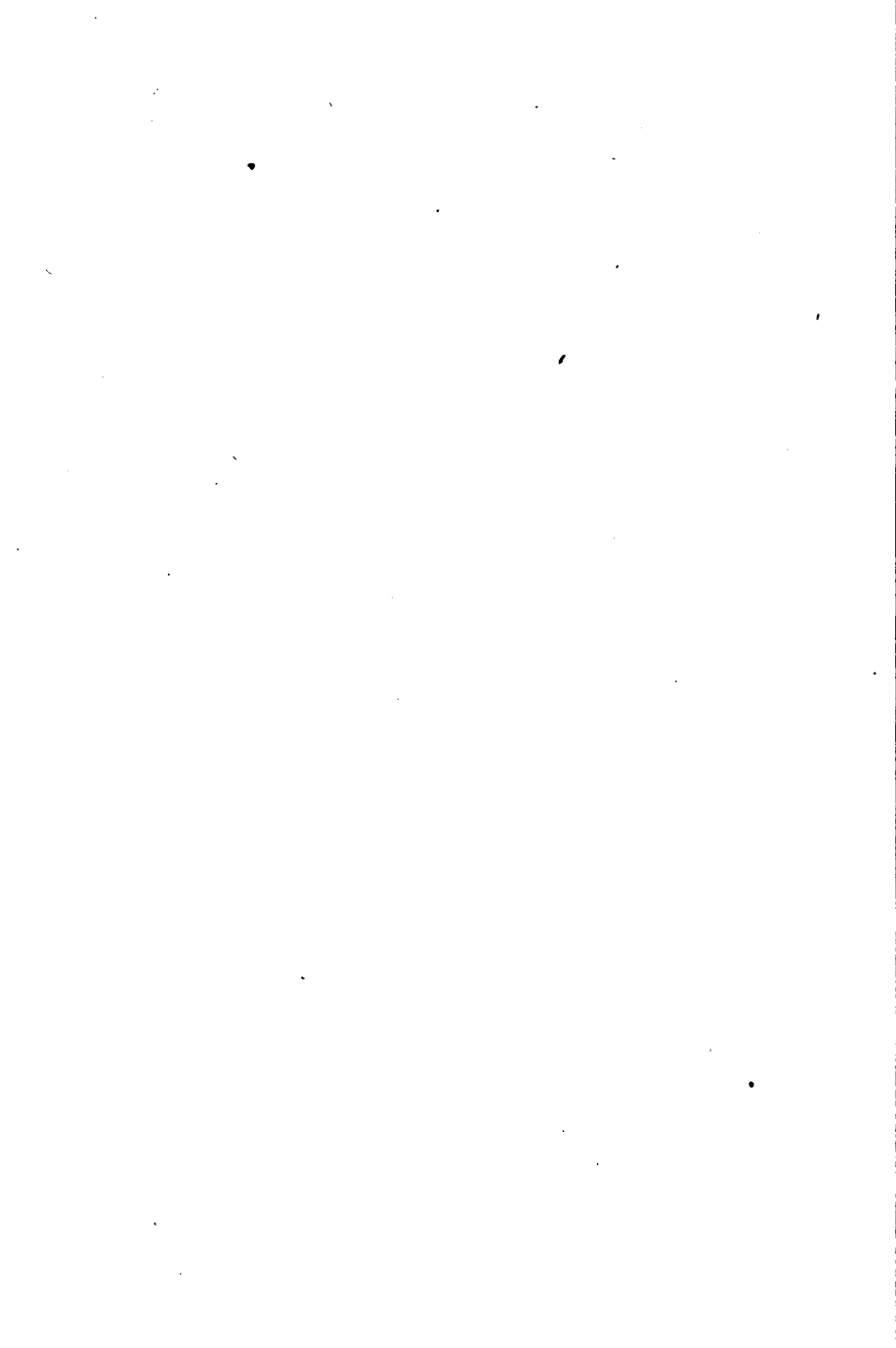
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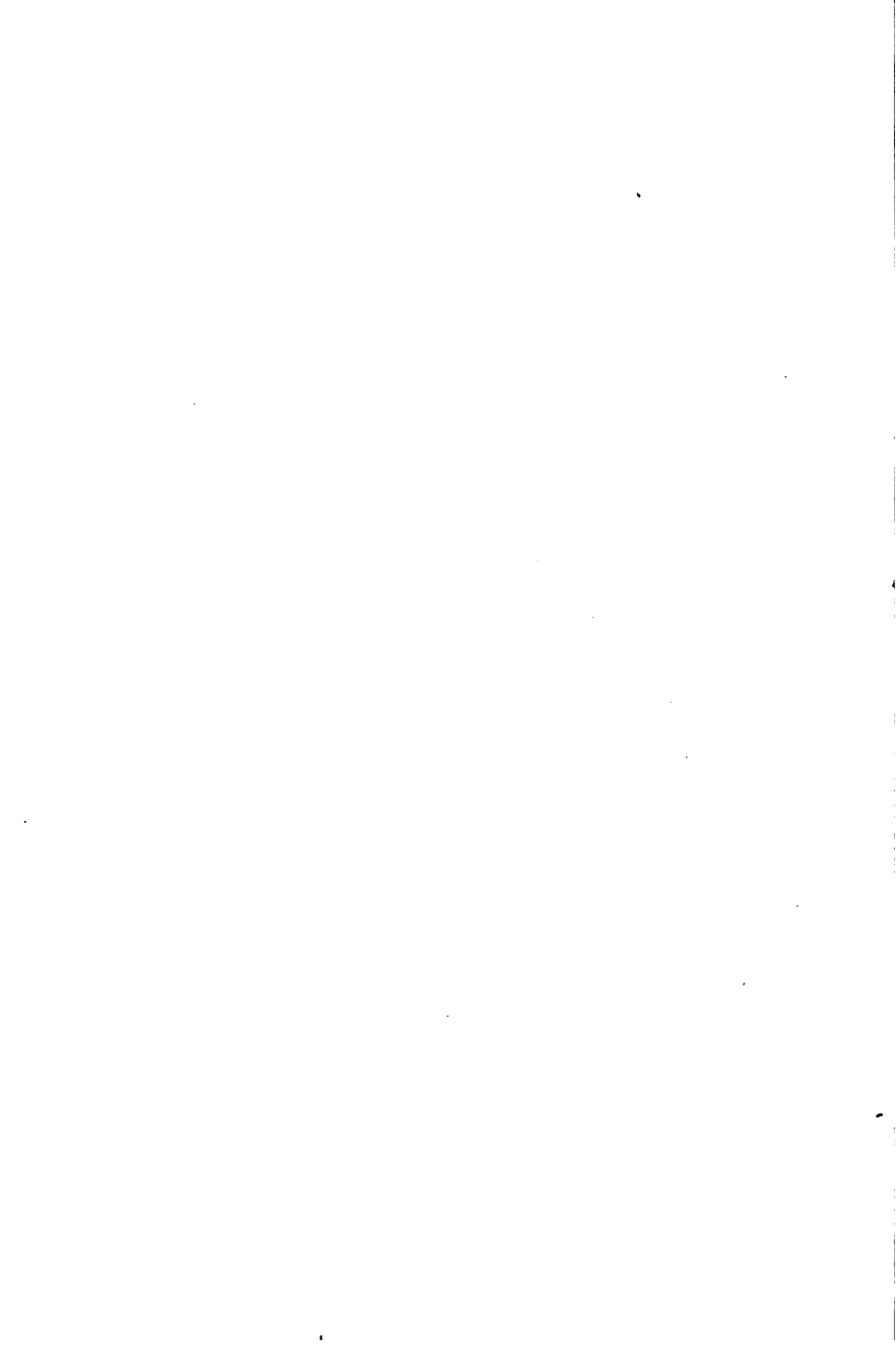
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**COMMON AFFECTIONS
OF THE LIVER**



COMMON AFFECTIONS OF THE LIVER

BY

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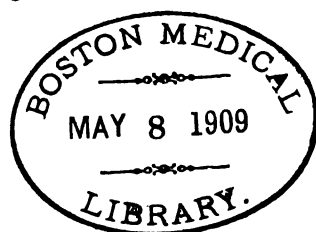
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PREFACE

So many excellent accounts of diseases of the liver have been written that it is necessary to apologise for and explain the appearance of this little book. I have from time to time been asked to reprint some of my clinical lectures, but I have never done it. Then I was asked to write a short book on the clinical aspects of diseases of the liver; so I have, after much persuasion, taken my notes of such of my clinical lectures as have dealt with diseases of the liver, and have arranged them so as to form this book. It represents clinical teaching. The rarer conditions are only mentioned briefly, the excessively unusual are omitted altogether. It is intended for students beginning their work, and not for those who have extensive clinical knowledge. They will find all they need in Rolleston's "Diseases of the Liver, Gall Bladder, and Bile Ducts."

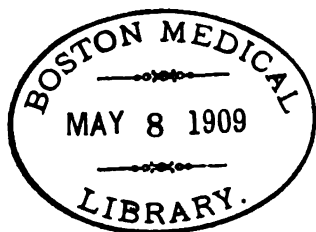
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COMMON AFFECTIONS OF THE LIVER

ANATOMICAL CONSIDERATIONS

THE liver in health is about $\frac{1}{8}$ of the weight of the whole body, or to be more accurate, it weighs between 45 and 60 ounces. At birth it weighs $\frac{1}{18}$ to $\frac{1}{24}$ of the body weight, but gradually the growth of the body outstrips that of the liver. It is of considerable importance to remember that in childhood it is relatively large.

It occupies the right hypochondriac and epigastric regions, and often extends into the left hypochondriac and right lumbar. On deep inspiration, in thin people whose abdominal muscles are lax, the lower edge of the right lobe, which is that between the anterior and under surfaces, can be felt by the fingers to descend if they are thrust up under the ribs, for in the supine position the right subcostal margin from the outer border of the rectus to the top of the last palpable rib marks the lower margin of the liver. In the upright position it may descend a half to one inch below this. In the epigastric

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angle a small portion of the anterior surface of the left lobe comes in contact with the anterior abdominal wall; often this cannot be felt owing to the rigidity of the recti; but, on the other hand, it may be the only part that can be felt when the ribs are raised, as they are in emphysema. The highest part of the upper limit of the liver may be indicated by a horizontal line which crosses the body at the junction of the sternum with the ensiform process—the sterno-ensiform line. This line indicates the upper margin of the liver at the junction of the fifth rib with its costal cartilage—that is to say, in the mid-clavicular or nipple line. In the middle line the upper border of the liver crosses the middle of the ensiform cartilage. From its highest point in the nipple line the upper margin of the liver descends to the seventh rib in the mid-axillary line. The hepatic dulness to the left of the sternum cannot be distinguished from that due to the heart. On the right it begins at the middle of the ensiform process of the sternum; in the nipple line it reaches the upper part of the fifth intercostal space, in the mid-axillary line the seventh, and in the line of the scapula the ninth; but its limit at the back is often difficult to determine precisely. The exact relationship of the liver to the abdominal wall varies slightly in health, for the organ is often a little lower in the erect than in the horizontal posture, the upper and lower margins both often dropping

half an inch or a trifle more; and it descends on inspiration and ascends on expiration. It must be borne in mind that although the sterno-ensiform line crosses the fifth rib in a healthy subject, it crosses the fifth space in emphysema, and the fourth space if the ribs are abnormally depressed. In the upright posture the liver rests on a shelf formed by the right kidney, colon, stomach, first part of the duodenum, gastro-hepatic omentum, pancreas, and coeliac axis. Behind, it is fixed by the hepatic veins and inferior cava, connective tissue, and peritoneal reflections.

Much lung intervenes between the liver and the skin, and it is often of importance to remember that posteriorly the thin lower border of the lung does not reach lower than the tenth rib, so that below the tenth rib the costal and diaphragmatic portions of the pleura are in contact; and that the inferior limit of the pleura does not quite extend to the attachment of the diaphragm, but leaves a small portion of the circumference of this muscle in contact with the costal parietes. The fact that the superior surface of the liver has on it a slight depression made by the heart is of no importance, for, owing to the strength of the fibrous part of the diaphragm, alterations in the heart do not directly affect the liver; but the close contact of the left lobe of the liver to the lesser curvature of the stomach, and the contact of the pylorus with the quadrate lobe, are of great

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importance, for not only may ulcers and malignant disease of the stomach grow directly into the liver, but tumours of the stomach may sometimes be dragged up and down during respiration, moving with the liver. For the same reason tumours of the hepatic flexure of the colon or of the kidney may move up and down with respiration, as both these structures are adherent to the inferior surface of the liver.

Turning to the liver itself, very rarely the organ is lobulated so as to suggest a resemblance to foetal lobulation of the kidneys. The cause of this is not known; probably it is not a congenital defect, but a result of some past disease, new-formed fibrous tissue causing deep indentations of the liver.

When the viscera are transposed the right lobe is small, and the left large if the liver is transposed, as well as the other viscera. Very rarely this alteration is seen, even when the organ is not transposed. It is then generally the result of syphilitic disease, leading to shrinking of the right lobe. Sometimes from the same cause the left lobe may be very small; sometimes it is dwarfed without any apparent cause. It is not uncommon to find small projections of the liver substance; usually they are on the under surface of the right lobe; they are of no significance. Isolated fragments of hepatic tissue have been found in the suspensory ligament, but they are very rare.

A tongue-like projection of the right lobe may

protrude from its lowered right-hand part. This projection, which is called Riedel's lobe, is often associated with disease of the gall bladder, such as gall-stones, or with tight-lacing, and is commoner in women than in men; but it may be found in quite young children, so sometimes it must be regarded as an anatomical abnormality. The connection between a Riedel's lobe and the liver may be only peritoneum, in which case the lobe may easily be confounded with a floating kidney, especially as there may be a band of resonance between it and the liver. A Riedel's lobe may also be confused with almost any abdominal tumour which occurs on the right side of the abdomen; it rarely gives rise to any symptoms, but is often associated with those of gall-stones. It is not sufficiently recognised that it is often very difficult when palpating the abdomen to tell the lower right-hand part of the right lobe from the right kidney, even when there is no projection that can be called a Riedel's lobe.

Many conditions unconnected with the liver cause an apparent alteration in it. Thus, when the body is wasted and the tissues are lax, the organ may drop down—further reference will be made to this presently when describing floating liver—but, quite apart from this condition, a general weakness of tissues will lead to its dropping; thus I have twice known men who were wasted from diabetes thought to be suffering from cancer of liver because that organ

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was believed to be enlarged, as indeed it may be in diabetes, but in these two cases it had probably only dropped down in the abdomen. In neither case had the urine been examined. Further, when the liver is considerably enlarged and heavy it may drop, and it is no uncommon thing to find that the nutmeg liver of the patient with heart disease who sits propped up in bed has dropped a little. This is ascertained by noticing that the upper limit of the hepatic dulness is lower than it should be, and of course the liver is not as large as mere palpation appears to indicate, although it must be remembered that it can increase considerably in size and weight from mere vascular distension, for in severe congestion—*e.g.* in mitral disease—its weight may increase by two-thirds.

Then alterations in the chest lead to depression of the liver, which may erroneously be thought to be enlarged. Thus in an extreme case of fibrosis of the lungs with adherent pleura, I have seen the sucking in of the ribs on inspiration lead to depression of the liver down to the umbilicus, and the right lobe may be depressed into the right loin by tight lacing; this is often associated with displacement of the right kidney. Again, the deformities of the chest, due to rickets or curvature of the spine, may lead to great depression of the liver. It is true that it may be depressed by large collections of fluid in the right chest, but when we remember

that three pints of fluid not infrequently collect in one pleural cavity and sometimes more—I have known ten pints—the liver is not so much depressed as might be expected, the reason being that with its firm attachments it is not easy to depress the diaphragm, and the fluid more readily makes room for itself by compressing the lung and pushing over the heart. When there is a very large amount of pus in the right pleura the liver will be more readily depressed than by simple fluid, for in a case of empyæma the diaphragm itself becomes inflamed, and so weakened. The liver may be depressed by a right-sided pneumo-thorax. If in diaphragmatic pleurisy the diaphragm is not working and is more or less in a constant position of inspiration, the liver is also constantly in this position, thus it seems a little depressed in some cases of right-sided pneumonia. We are usually told that extreme pericardial effusion will depress the liver, but I think this must be rare; certainly we should expect it to be, for the pressure would have to act through the fibrous part of the pericardium, which is very firm. It is often assumed that a subdiaphragmatic abscess being below the diaphragm will depress the liver considerably, but this is incorrect, for the numerous adhesions in connection with a subdiaphragmatic abscess generally prevent depression of the liver.

Artificial constriction of the thorax leads to

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changes in liver. Often quite a slight degree will cause the formation of furrows on the liver evidently corresponding to the ribs. But we may see furrows the direction of which is such that they cannot be due to the ribs; it has been suggested that they are due to the fact that the liver is compressed during inspiration and expands again during expiration. Tight-lacing may, by local constriction, lead to the formation of such a deep furrow that part of the liver is, during life, apparently separated from the rest. Recently a man was in the hospital with what was thought by many who saw him to be an elongated tumour lying transversely in the abdomen, but it was nothing but the lower part of the liver nearly cut off from the rest by the pressure of a belt he had worn. It is clear that the position of these furrows will depend upon the position of the pressure caused by tight-lacing. The furrow may be so deep that the blood supply of the isolated portion of the liver is impaired. Thus I have seen fatty change confined to such a part, and others have described cirrhotic and syphilitic changes and new growth restricted to it.

The effect of corsets or other artificial pressure upon the liver as a whole will depend upon the direction of the pressure; most commonly the liver is forced down, flattened and elongated from above downwards. The liver thus forms an apron, covering

much more of the abdominal viscera than is natural, but some of the intestines may get in front of it. Such pressure often leads to a transverse depression across the right-hand lower part of the right lobe, so that a more or less detached piece of liver lies over the right kidney, and is, indeed, an artificial Riedel's lobe (see p. 5), and may lead to the same difficulties of diagnosis. Less commonly the liver is pushed upwards, with the result that it is shortened in its vertical diameter and elongated laterally, so that the left lobe may touch the spleen. Women suffering from any of these conditions should be advised to wear a straight-fronted corset such as that described later (see p. 18).

The protrusion of the liver through the diaphragm (diaphragmatic hernia) is very rare, for diaphragmatic hernia is usually the result of traumatic laceration of the diaphragm, and this hardly ever takes place on the right side, because the bulky liver supports the diaphragm and prevents its being torn.

Various abdominal conditions alter the position of the liver or the ease with which its size may be ascertained. It is quite rare for enlargement of the liver to lead to any upward extension of the hepatic dulness. This is what would be expected, for the mere weight of the enlarged liver will lead to its falling, and the resistance of the intestines and abdominal walls is much less than that of the diaphragm, so the enlarged liver will grow in the

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direction of least resistance. Raising of the hepatic dulness upward is best seen when some local disease of the liver directly implicates the diaphragm; thus an abscess of the liver will grow from the upper surface of the liver, soften the diaphragm, and extend upwards; a hydatid may do the same. From this it follows that usually when there is extension upwards of the hepatic dulness, it is a local extension, forming a dome-shaped addition to the hepatic dulness. It is conceivable that a mass of malignant disease in the liver might lead to an irregular extension up of the hepatic dulness; but such an event must be exceedingly rare, for malignant disease usually affects the whole liver, and so the organ enlarges downwards. Very large collections of ascitic fluid or very large abdominal tumours may push the liver up, and so lead to an increase of hepatic dulness upwards; but this is not common, for such tumours or collection of fluid will more readily cause protrusion of the anterior abdominal walls and compression of the intestines. A subdiaphragmatic abscess (see p. 114) on the right side, because of the adhesions between the liver and abdominal wall which it causes, rarely pushes the liver down, but by its addition to the hepatic dulness it will cause a localised dome-shaped increase of hepatic dulness upwards. Such an abscess is usually due to an appendix abscess tracing upwards in front of the right kidney or to a gastric ulcer.

Tumours at the back of the abdomen, such as those of the pancreas or large aneurisms, may push the liver forwards.

There are three moderately common tumours which lie transversely in the abdomen, and so may be thought to be the lower margin of an enlarged liver. They are: a stomach affected with malignant disease, especially when the growth infiltrates much of the greater curvature; malignant disease of, or impaction of fæces in the transverse colon; and the great omentum thickened and puckered up towards the transverse colon by some form of chronic peritonitis. Any of these tumours may move up and down with respiration, for they are all directly or indirectly attached to the liver; but the movement is not usually so extensive as that of the liver should be, and a band of resonance may sometimes be detected between the liver and the tumour, or the edge of the liver may be felt above it. Enlargements of the pylorus and thickening in connection with a gastric and duodenal ulcer may all be difficult to tell from an enlarged gall bladder, not only from their position, but because they may move up and down with respiration. We have already mentioned that it is not always easy to tell a floating kidney from a hepatic tumour. It is very rare for the adhesions of chronic peritonitis to drag the liver out of place, but it has been known to be pulled downwards.

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The hepatic dulness may be altered by gas. Thus it may be almost obliterated by the descent of an emphysematous lung, and a slight lowering of the upper margin of the hepatic dulness from this cause is quite common. Another result of emphysema is that the lower ribs stand out so much in a position of extreme inspiration that they bring the upper part of the abdominal wall far forward, and therefore it is often quite impossible to feel the liver.

When, as in perforative peritonitis, free gas is present in the peritoneal cavity, the gas, getting in front of the liver, may diminish the hepatic dulness, but this sign is often absent. On the other hand, obliteration of the lower part of the hepatic dulness by no means always indicates that there is free gas in the peritoneal cavity, for coils of intestine or the colon may get between the liver and the anterior abdominal wall. This is of no clinical significance. Very rarely diminution of the hepatic dulness is due to shrinking of the organ; acute yellow atrophy is an instance in point. A large collection of ascitic fluid may render it difficult to estimate the size of the liver.

HEPATOPSIS OR WANDERING LIVER

THESE terms may be applied to a liver which, being unduly displaceable, leaves its normal position and forms an abdominal tumour. The phrase movable liver is sometimes used. Floating liver is a bad name, for normally the liver floats in the abdominal cavity. Floating kidney is a correct phrase to apply to certain abnormalities of position of the kidney, for it is in health fixed at the back of the abdomen. It is to be particularly noticed that a liver displaced by a tumour, or any other cause, is not a wandering liver, for it is probably just as fixed as a normal liver; but a wandering liver can be easily moved by manual pressure or alteration of position.

Much confusion has arisen from the use of the unfortunate phrase "partial hepatopsis." It has been applied to a Riedel's lobe or to pieces of liver more or less cut off from the rest of the organ by wearing bands or belts. It should be discarded, for it has nothing to do with the condition now under discussion; but it is worth remembering that sometimes the part of the liver cut off from the rest by wearing a band has been thought to be the whole liver, and

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has been considered to be an example of hepatopsis. It is much more common to find a wandering liver in women than in men ; most of the patients are over forty. There are various degrees of hepatopsis ; often the organ is only slightly displaceable. Extreme degrees are certainly rare and are often part of a general visceroptosis, in which not only the liver but also the kidneys, spleen, stomach, and bowels are displaced.

It is frequently difficult to explain how a liver becomes movable ; generally it appears to be because the abdominal walls are loose and pendulous, so that the natural support which they give the liver by means of pressure applied through the intestines is gone, for there is no doubt that the liver and other viscera are chiefly kept in place by the contraction of the abdominal muscles, the transversalis and obliques are especially powerful in this. It has been suggested that in badly nourished and anæmic women the ligaments of the liver lose their tone, and hence the organ drops ; and this may be so, for Keith has shown that they contribute considerably towards keeping the liver in place. Very likely the same cause that leads to weak abdominal walls will induce weak ligaments. Tight-lacing leads to weakness of the abdominal muscles, and it also pushes the liver down. Variations in the atmospheric pressure within the abdomen have been thought to be the cause of the alteration of the position of the viscera, but this is quite erroneous.

Extreme degrees of hepatopsis are called total hepatopsis—a bad name, for the liver is not displaceable to an indefinite extent. When the condition is extreme the organ rotates on a transverse axis, so that the upper diaphragmatic surface comes in contact with the abdominal wall, and the anterior surface points a little downwards, the result is that a large amount of liver comes in contact with the anterior abdominal wall. At the same time the liver rotates a little on a vertical axis, so that the convexity of the liver is turned to the right and the under-surface to the left. The result of all this is that when the liver is examined it is found to have fallen and to be flattened, extending down to the umbilicus, and with the most prominent part of it near its lower part and on the right. Often the depression downwards is increased by tight-lacing, and this may lead to a groove across the upper part of the organ. The displaced liver may form a slight protrusion of the abdominal walls; it is easily palpable, moves up and down with respiration, can usually be pushed back into its normal position when the patient lies down; indeed, sometimes on lying down it goes back of its own accord, only to fall again when the patient stands up. It is movable also laterally, and can with the hands be rotated about a horizontal axis passing through the attachment of the organ to the inferior cava, this being always the most fixed point of the

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liver, but when the liver drops much, the transverse fissure may descend two inches. This must throw great strain on the hepatic vessels and impair the blood supply of the organ. Sometimes it rotates about a vertical axis when the patient turns from side to side. If it has dropped considerably, so that the greater bulk of it is well down in the abdomen, there is considerable diminution, or even absence, of the hepatic dulness that should be present over the ribs; the hand may, indeed, be passed up between the liver and the ribs, and at the upper part of the abdomen on the right side there is a depression of the anterior abdominal wall. This is very characteristic. Whenever the liver drops considerably the diaphragm falls too and its fibres are shortened.

There may be no symptoms, but the patient usually complains of a dragging pain and sensation of heaviness in the hepatic region. This is naturally much worse in the erect position, with the result that in a bad case the patient has always to lie down. Sudden severe attacks of pain in the hepatic region are very common. These may be due to gall-stones, which are not infrequent in those who suffer from hepatosis, or to the presence of a floating kidney. When neither of these causes are operative, it may be caused by kinking of the cystic or hepatic duct dependent upon the displacement of the liver, and naturally, therefore, sometimes jaundice is

HEPATOPSIS OR WANDERING LIVER 17

associated with a movable liver. The patients are often dyspeptic, neurotic valetudinarians. In about a quarter of them a floating kidney may be found, the stomach may be dropped, and if the abdominal walls are very thin intestinal peristalsis may be visible; rarely membranous colitis is present. In health the muscular wall of the abdomen by its contraction prevents stagnation of blood in the abdominal vessels when the patient assumes the erect posture. Most patients afflicted with a wandering liver have very weak abdominal muscles; hence they suffer much from faintness, dyspnoea, palpitation, and exhaustion on exertion, but these symptoms pass away when the patient lies down. Many mistakes in diagnosis have been made, but usually because the possibility of hepatopsis has been forgotten.

The first point in the treatment is to improve the tone of the abdominal muscles. This may be done by systematic exercises every morning. It is a good thing for the patient to lie on the floor on her back and sit up while keeping the arms folded. This obviously exercises the abdominal muscles, and should be repeated several times. When the patient is on her back it is well to flex the thighs several times on to the abdomen. Deep breathing also employs the muscles, and daily abdominal massage is very useful. Blocks under the foot of the bed raise the hips higher than the chest, so that when the patient is in bed the viscera

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fall towards the diaphragm. Much benefit follows the wearing of a properly designed corset; it should have a firm grip on the ilia, be loose at the upper part, and be so made that by lacing it from below upwards considerable pressure is brought to bear on the lower part of the abdomen; it should be laced up when the patient, in the erect posture, drawing a deep inspiration, thus raises the ribs and at the same time contracts the abdominal muscles as much as possible. This movement drags and forces up the viscera, and the lacing of the corset thrusts them up still further and keeps them in place. Some authorities advise strapping the abdomen with strips of wide strapping. Each strip should be cut so as to be about four inches in width one end and seven the other, and should reach from a point about four inches from the middle line past the middle line in front round to the spine. The foot of the bed should be raised and the first strap put on at the level of the pubes, the next a little higher on the opposite side, and so on alternately till the whole of the front of the abdomen is covered with strapping very tightly applied, the anterior end being fixed first. Suitable rubber strapping is known as "30" plaster—it contains zinc oxide—and the abdominal wall should be well cleaned and, if necessary, shaved before it is applied. As far as my experience goes, strapping the abdomen is neither so efficient nor so comfortable

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as the corset. The attempt has been made to stitch the liver in place, but the operation often fails, and if pressure is properly applied it is rarely, if ever, needed. There is nothing about the treatment of the dyspepsia or neurasthenia that calls for special notice.

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JAUNDICE

A PERSON is said to suffer from jaundice when sufficient bile pigment circulates in the blood to stain the skin or conjunctivæ yellow. The word is derived from the French *jaune*, yellow. The word icterus is employed by some writers in place of jaundice; its derivation is uncertain. It must be clearly understood that jaundice is only a symptom. When we see a patient who is jaundiced two problems always present themselves: firstly, why has he bile pigment in his blood? secondly, what effects is this bile pigment producing?

The presence of bile in blood is, in more than four-fifths of all cases of jaundice, due to an obstruction to the flow of bile in the ducts, which would be obvious if the patient died there and then. The bile continues to be secreted, but as its natural exit is closed, it is taken up by the lymphatics, and so passes into the general circulation. That this is the explanation of jaundice due to obstruction to the exit of bile has been shown by many experiments, *e.g.* jaundice does not occur as a result of ligaturing the common bile duct if the thoracic duct is also tied. When the obstruction has lasted a long

while, the larger bile ducts behind the obstruction contain only clear mucus; this has been secreted by the mucous membrane lining the bile ducts, and the bile has been taken up by the lymphatics from the smaller ducts before it has had time to reach the larger, because the obstruction is sufficient to raise the pressure of the bile in the bile ducts considerably above its usual low point. The conditions which cause obvious obstruction to the exit of bile into the duodenum are as follows. They will be placed roughly in the order of their frequency in actual practice.

Catarrhal Jaundice.—This is almost certainly due to a catarrhal inflammation of the duodenum spreading into the common bile duct.

The reasons for this belief are that the clinical course of the disease is such as would be produced by an inflammation of the mucous membrane of the common duct, the appearance of jaundice is often preceded by symptoms of gastro-duodenal catarrh, and in some of the few cases in which death has occurred during an attack of catarrhal jaundice, the mucous membrane of the common duct has been found swollen, or a plug of mucus has been discovered in it. If we remember the very small size of the opening of the common duct into the duodenum, it is easy to understand that swelling of its mucous membrane or the presence of a plug of mucus will prevent the exit of some, at least, of the bile. It is no doubt true that sometimes nothing

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can be found after death to explain a case of catarrhal jaundice, but it must be borne in mind that alterations in mucous membranes are usually difficult to recognise at a post-mortem examination.

In many cases the symptoms of the gastro-intestinal catarrh, which causes the catarrh of the common bile duct, are evident. Thus for a few hours, or even a few days, before the onset of the jaundice the patient complains of a sense of heaviness, or even actual pain, over the stomach; his tongue is furred, his appetite is poor, he dislikes the idea of taking food, he feels sick and may perhaps vomit, the bowels are constipated, and the urine is high coloured and contains an excess of lithates. There is often a slight rise of temperature, the pulse is quickened, the patient feels out of sorts and complains of a headache. The severity of these preliminary symptoms of gastro-intestinal catarrh varies, and they may be hardly evident. Soon the patient becomes yellow, for the free exit of the bile into the duodenum is hindered, and hence bile is re-absorbed from the biliary passages by their lymphatics and passed into the blood. He then suffers from such of the clinical symptoms due to the circulation of bile in the blood (see p. 33) as do not require the prolonged presence of bile there for their production; for example, his conjunctivæ and skin remain bright yellow, the skin does not become dark green, and he does not show patches of xan-

thalamas, and he does not become comatose. His urine contains bile, the motions are pale, but they are nearly always somewhat coloured, because the obstruction to the outflow of bile is rarely complete. Itching is often very troublesome, and what with the gastro-intestinal catarrh, and what with the bile in the blood, these patients, who are usually young subjects and more commonly males than females, suffer much from depression of spirits. The distaste for food, the headache and the constipation now become greater, the temperature remains raised, or, if not up before, mounts a little, it is but seldom that there is enough bile in the blood to reduce the rate of the pulse, the liver may be slightly enlarged from engorgement with bile, and the distension of it may induce some pain and tenderness. Generally in four or five days—but sometimes more, sometimes less—after the jaundice appears the patient begins to feel better, and he slowly recovers; the bile in the urine becomes less, that in the motions increases, but it is often a fortnight or more before the staining of skin and conjunctivæ disappears. Some persons are liable to suffer from recurrent attacks of catarrhal jaundice. A plug of mucus at the end of the common duct must block the pancreatic duct unless it open separately, but we do not know any symptoms due to such a block. Catarrhal jaundice is of itself never fatal, and therefore we can only

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investigate the morbid anatomy of it when a patient suffering from it chances to die from some accident or other disease during his course of it.

The patient should be put to bed; he is so depressed that he willingly obeys this order. He should take no solid and not much liquid food. Peptonised milk and thin beef tea will suffice. It is often a good thing to drink abundantly of hot water, and the bowels should be thoroughly opened. It is best to begin with a good dose of calomel, say three or four grains for an adult, and then to keep the bowels open with a daily dose of any saline aperient, such as Epsom salts or phosphate of soda.

Gall-Stones.—These frequently obstruct the common bile duct and cause jaundice, not only because they stop the exit of bile, but because they set up some cholangitis, and the consequent swelling of the lining membrane of the smaller ducts leads to obstruction of them with resulting absorption of bile. Gall-stones do not come within the scope of this book, but are described in Mr. Bland Sutton's "Gall-Stones and Diseases of the Bile Ducts."

Malignant Disease of the Liver (see p. 213).—In this disease the jaundice is usually due to the pressure on the common bile duct of glands in the portal fissure which have become enlarged from the deposition in them of malignant growth, because the liver is affected with malignant disease; but sometimes the pressure is exerted on the larger ducts

in the liver directly by the malignant disease of the organ.

Cirrhosis of the Liver (see p. 127).—The jaundice is caused partly by the increased fibrous tissue exercising compression on the smaller ducts, and partly by the fact that probably there is often some swelling of the mucous membrane of them.

Carcinoma of the Head of the Pancreas.—The pressure of the growth on the terminal part of the common bile duct leads to jaundice, and naturally jaundice follows in most cases in which the growth is in the head of the gland. Jaundice is present in between a half and two-thirds of all cases of carcinoma of the pancreas. Primary malignant disease of the pancreas was found in 31 out of 6708 persons on whom in fourteen consecutive years post-mortem examinations were made at Guy's Hospital. It is rare under forty, and over sixty-five years of age. The disease is usually rapid, for disease of such an important gland interferes much with digestion. Severe abdominal pain and tenderness are both very common; often tenderness is present. The patient is usually anæmic and wastes considerably. In a considerable number of cases the enlarged head of the gland can be felt as a tumour, and this would oftener be possible were it not for the presence of secondary growths in the liver or ascites, which occurs in about one-third of the cases. The muscles over the growth are often rigid. The stools may be

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fatty. It is often said that when the common bile duct is obstructed as a result of malignant disease, the gall bladder is usually dilated from distension by bile and mucus, and that when the obstruction is due to gall-stones the gall bladder is frequently contracted because the gall-stones which were there before they caused jaundice have set up chronic inflammation of its walls. This is known as Courvoisier's law. No doubt, speaking generally, it is true, but often the conclusion is hastily drawn that in all cases of obstruction by growth the gall bladder is dilated; that certainly is not so, for although it may be dilated when growth of the pancreas causes jaundice, it often is not, and even if it is the enlargement of the liver frequently makes it difficult to feel during life.

Chronic Pancreatitis.—Every one with a large experience in the post-mortem room will admit that chronic pancreatitis leading to jaundice is rarely fatal. Nearly every fatal case in which during life this diagnosis is made turns out to be an example of malignant disease of the pancreas, as may be proved either by the occurrence of secondary deposits or by histological examination of the pancreas. Looking through the records of 19,000 post-mortem examinations I only came across one fatal case of undoubted chronic pancreatitis with jaundice. The patient, a man aged forty-two, was jaundiced for seven months; he had an enlarged liver, gradually became weaker,

and vomited much towards the end. The head of the pancreas was hard and much enlarged; the pancreatic ducts and bile ducts were much dilated. The pancreas, when histologically examined, only showed an increase of connective tissue. The enlargement of the liver was due to the obstruction to the bile duct. Nevertheless it is probable that chronic pancreatitis is a cause of jaundice in a moderate number of cases, for it is not infrequent to meet with cases of chronic jaundice not due to growth, almost certainly not due to gall-stones, and lasting too long to be catarrhal. The belief that some at least of these cases are due to chronic pancreatitis is derived from the fact that when surgeons have opened the abdomen, the pancreas has been felt to be hard and enlarged. Often the gall bladder has been drained in these cases, but as the disease is rarely fatal if left to itself, it must tend to slowly get well, and therefore surgical interference is not really needed, especially as those who are deeply jaundiced bear operations badly. All those with a large experience must have met with patients in whom chronic jaundice, after lasting several months, has slowly disappeared.

Passive Hyperæmia of the Liver.—This is best seen in severe cases of over-distension of the venous system following upon mitral disease. The dilated venules in the liver substance press upon the minute bile ducts, and so lead to an absorption of bile.

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If we except the jaundice of the newly born (see p. 296), which in most cases can hardly be called a disease, the above are the only causes of jaundice that are usually met with in temperate climates. It is very important to remember this at the bedside, for the causes of jaundice are so numerous as to be bewildering to the student, and enough to make him despair of ascertaining the cause of a case of jaundice with any certainty; but, as a matter of fact, it is not usually difficult, for most cases of jaundice can be assigned to one of the causes just mentioned.

The remaining causes—all quite exceptional—of jaundice which lead to obvious obstruction to the exit of the bile are: an abscess or a hydatid cyst of the liver may press on the bile duct; carcinoma or cicatrising ulcer of the duodenum may constrict its duodenal orifice; a large abdominal tumour, such as a huge gastric carcinoma, a tumour of the hepatic flexure of the colon, a pancreatic cyst, an ovarian or uterine tumour, a retroperitoneal tumour, an aneurism (for hepatic aneurism, see p. 73), or a supra-renal or renal tumour may compress the common duct. A few cases have been recorded in which a floating kidney caused jaundice. I have seen one. The patient was a man, and if his kidney, which was very movable, was pressed up towards the liver and held there, in an hour or two bile appeared in the urine. His kidney was stitched in place, and after this was done he never had any jaundice. Gum-

mata of the glands in the transverse fissure and gummata of the pancreas, tuberculous disease of the glands in the transverse fissure, a large pancreatic calculus, and adhesions in connection with an old gastric ulcer have all been known to give rise to jaundice by pressure on the bile duct, but so rarely that the occurrence of jaundice in these conditions is a mere curiosity. Considering that a pyloric gastric ulcer and an ulcer of the first part of the duodenum are common, and that the duodenum and pylorus are in contact with the liver, it is strange that adhesions dependent upon these ulcers so rarely cause jaundice; indeed, they hardly ever do so, and the thickening of the capsule of the liver which occurs in perihepatitis rarely, if ever, leads to jaundice. It has been said that the bile duct may be kinked in association with gasteroptosis or a wandering liver, but this is doubtful; but it must be remembered that gall-stones are frequently associated with hepatoptosis (see p. 16). Jaundice has been known to follow rupture of a hydatid cyst into the bile ducts, or to be due to the presence in them of various species of *Distoma*, or to be caused by an ascaris lumbricoides entering the common bile duct; but in this country at any rate these causes are mere curiosities. Simple stricture of the bile ducts and primary growth of them in rare instances lead to jaundice; but they are described in Mr. Bland Sutton's "Gall-Stones." There seems no doubt that

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jaundice may be due to severe emotion; thus the case has been described of a man who became jaundiced just before fighting a duel, and I have heard of the case of a child who became jaundiced when she saw her sister accidentally drowned. This variety of jaundice has been ascribed to spasm of the bile duct, but this explanation is doubtful.

Jaundice not dependent upon obvious obstruction to the flow of bile is of far less importance. Formerly it was thought that such jaundice might be due to the formation of bile pigments in the blood from hæmoglobin; sometimes from some fault in the blood—hence the phrase hæmatogenous jaundice—sometimes because the liver cells had struck work—hence the term jaundice by suppression—and so the bile pigments were formed in the blood instead of in the liver. But now it is believed that bile pigments cannot be formed elsewhere than in the liver, for it has been shown that poisons (*e.g.* arseniuretted hydrogen), which cause jaundice without obvious obstruction to the ducts, and were therefore thought to do so by the formation of bile pigments from hæmoglobin in the blood, do not cause jaundice if the liver is extirpated or its vessels are ligatured. Another suggestion, made to explain jaundice not due to obvious obstruction, was that there was such a free secretion of bile that the excess was absorbed from the intestine, and hence jaundice. The belief at the present day is that none of these

explanations hold good, and that all jaundice is obstructive; but that when the obstruction is not obvious, it is in reality due to swelling of the mucous membrane of the minute ducts and the secretion of very tenacious mucus by them, with consequent obstruction of them. This has actually been observed as a result of some poisons, *e.g.* toluylendiamin, which cause jaundice without obvious obstruction of the large ducts. The outflow of bile is thus prevented, and absorption of it by the lymphatics takes place. Therefore all jaundice is obstructive. Inasmuch as the swelling of the mucous membrane of the minute ducts in these cases in which the obstruction is not obvious is always due to irritation produced by poisons—usually bacterial—this variety of jaundice is best called “toxic.”

It so happens that many poisons which cause toxic jaundice, *e.g.* toluylendiamin, phosphorus, that of severe pyæmia, also cause much destruction of the red cells of the blood; but destruction of them does not lead to jaundice unless there is also obstruction in the minute ducts to the outflow of bile.

Perhaps the commonest form of toxic jaundice is that associated with pyæmia. The primary focus may be anywhere, but jaundice is often seen when it is in the periphery of the portal system (see p. 52). Occasionally there is no obvious seat of infection, and a patient may suffer from pyæmia and deep jaundice, and may even die, and yet at the post-

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mortem examination nothing abnormal to the naked eye can be seen anywhere in the body; but a pure culture of streptococci may be recovered from the blood. Jaundice may be associated with pneumonia, malaria, and relapsing fever; but in these infections it is very rare and usually slight. On the other hand, it is very striking in yellow fever, Weil's disease, and acute yellow atrophy. Sometimes the swelling of the minute bile ducts is not due to poisons manufactured by micro-organisms. Thus it may be caused by phosphorus, snake-bite, or toluyldiamin, all of which lead to jaundice. Other poisons may lead to jaundice, but the cases in which they do so are so few that they are of no practical importance. Jaundice may be seen in paroxysmal hæmoglobinuria; here some product of the destruction of the blood is thought to set up inflammation of the minute ducts.

It is particularly to be noted that in all these toxic forms of jaundice in which the obstruction is not evident to the naked eye, the jaundice is not usually deep; it never becomes the dark green seen in long-standing jaundice, due to complete obstruction of the common bile duct. This is because it is rare for all the minute ducts to be obstructed, and for the same reason the fæces often contain some bile; indeed, occasionally a large amount, for the blood destruction so often associated with toxic jaundice, setting free a large amount of hæmoglobin, renders the formation of bile pigment by the liver so easy

that in spite of some ducts being obstructed, the total amount of bile secreted is increased.

Naturally the urine contains less bilirubin in toxic than in completely obstructive jaundice; but, on the other hand, as some bilirubin reaches the intestine and there urobilin is formed from it, the urine contains urobilin, which it does not in the completely obstructive form.

Clinical Symptoms due to the Circulation of Bile in the Blood.—The conjunctivæ are stained yellow, and it is here that jaundice is first seen. For the yellowness to be due to jaundice it must be present all over the conjunctivæ. Beginners are apt to mistake the yellowness of the masses of fat at the inner and outer canthus for staining by bile. No discolouration of the skin can be due to bile unless the conjunctivæ are stained yellow.

The skin becomes yellow a day or two after the conjunctivæ. The yellowness of the skin and conjunctivæ is due to bilirubin. The colour of the skin due to pernicious anæmia and chlorosis is liable to be mistaken for jaundice, but the mistake can easily be corrected by looking at the conjunctivæ. Slight jaundice is difficult to detect in those who are dark skinned, and often even moderately deep jaundice is overlooked when the patient is examined by artificial light. If jaundice lasts long it gradually becomes darker, and at the same time green from the formation of biliverdin; then the

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patient is said by the laity to suffer from black jaundice. It is well known that patients suffering from chronic jaundice bleed readily, and hence many small hæmorrhages are often seen in the jaundiced skin. Probably they are traumatic, trifling injuries producing visible hæmorrhages. When the cause of the jaundice passes away the staining of the skin fades very slowly.

The jaundiced patient frequently suffers from intolerable itching, and hence scratch marks are frequent. The cause of the itching is quite unknown; sometimes the patient complains of it before the jaundice appears; on the other hand, it may be absent when the jaundice is intense. It is often very difficult to treat successfully. The patient may be bathed all over with carbolic acid lotion of a strength of 1 in 40, or with an alkaline lotion consisting of 7 grains of sodium bicarbonate to the fluid ounce of water, or an alkaline bath of similar strength may be employed. A sixth of a grain of pilocarpine nitrate injected subcutaneously sometimes relieves it. Thyroid gland and antipyrin have both been tried. But not infrequently all these remedies fail, the patient's life is a perfect misery, and he cannot sleep. In extreme cases the only thing to do is to give a subcutaneous injection of morphia.

Xanthelasma, xanthoma, or vitiligoidea, as it was called by Addison and Gull, who first described it,

is a rare affection of the skin, which may occur in those who are not jaundiced, but which is seen in those who are jaundiced sufficiently often to indicate some special relationship between jaundice and it. It is a chronic deep dermatitis with early fatty degeneration; hence the patches of it are pale yellow. There are two varieties—one, the commoner—the *vitiligoidea plana* of Addison and Gull—consists of flat yellow patches, usually seen first on the upper eyelid just above the inner canthus. Later similar patches appear on the lower lids and other parts of the skin; they may be seen in the mucous membrane of the mouth, and Dr. Fagge records two cases in which the mucous membrane of the bile ducts was affected. The rarer form consists of nodules of similar colour (*vitiligoidea tuberosa*). They are usually seen on the ears and hands, especially on the extensor surfaces, and when they occur near a joint they look like deposits of urate of sodium.

It is often said that the sweat may be stained yellow, but the jaundiced skin is usually so dry that no sweat is secreted; however, in very rare instances the sweat in the axilla has been slightly yellow. The yellow staining of the nightshirt, often seen in those who are jaundiced, is due to urine, not to sweat.

The urine is very readily stained by bile, indeed, earlier than the skin; the principal bile colouring

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matter in the urine of a jaundiced patient is bilirubin, but there may be an excess of urobilin. The colour of the urine is dark, and varies from yellow to dark yellow or brown, greenish yellow and dark green, according to the duration and intensity of the jaundice. The later green tint is caused by biliverdin, bilirubin being converted into biliverdin by oxidising agents.

Inasmuch as urobilin, which exists in healthy urine, is formed in the intestine by bacterial action upon bilirubin excreted into the intestine in the bile, and is absorbed from the bowel and excreted in the urine, it is clear that when the obstruction causing jaundice is complete, so that no bile reaches the intestine, the urine will not contain urobilin after that formed in the gut has been completely absorbed and excreted, but in such cases there will be a great deal of bilirubin in the urine. If the obstruction is partial, some bilirubin reaches the intestine, and hence urobilin appears in the urine; and if, as is sometimes stated, bacterial action is especially active, the amount of urobilin may be considerable. Lastly, if the case is one of those just mentioned (see p. 32), in which while there is obstruction in only some of the smaller ducts, so that the patient is jaundiced, yet an excess of bile, and consequently of bilirubin, reaches the intestine, the amount of urobilin in the urine will be very considerable. The bile salts in the urine are con-

siderably diminished, or in many cases absent, after the first few days. The balance of evidence is that this is because, when a patient is jaundiced, the hepatic cells lose their power of forming bile salts. When the amount of bile reaching the bowel is diminished, the ethereal sulphates in the urine are increased. In some cases of jaundice the urine contains a little albumen and some epithelial casts, whether these are due to the presence of bile in the blood or to the disease of which jaundice is one of the symptoms is unknown.

When urine containing bile is shaken up, the froth is yellow. The clinical tests for the detection of bile in the urine are unsatisfactory, and will not, as a rule, show the presence of bile if the quantity is so small that it cannot be detected by sight. The following are in common use. Rosin's test: Let dilute tincture of iodine flow gently on to the surface of the urine in a test tube. A green ring forms at the line of contact of the two fluids if bilirubin is present, owing to its conversion into biliverdin by oxidation. Gmelin's test: If some fuming nitric acid—that is to say, nitric acid containing nitrous acid—is put into a test tube and urine containing bilirubin is allowed to flow on it, a series of coloured rings will form in the following order from below upwards—yellow, red, violet, blue, green. They are due to the oxidation of bilirubin, and the test is of no value unless the green ring is

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well marked. In Rosenbach's modification of this test, successive portions of the suspected urine are filtered through the same filter paper, and then a drop of fuming nitric acid is placed on the paper near the part wetted by the bile; as the nitric acid meets the bile the play of colours is seen. A common way of performing this test is to let a few drops of urine come into contact with a few drops of nitric acid on a white plate; if bile is present the play of colours is produced. The most reliable test is Huppert's. Barium chloride is added to the urine; the precipitate which carries down all the pigment is heated to boiling with alcohol and sulphuric acid by means of a water bath, and a green colour is produced if bile is present.

In the great majority of cases no other secretion than the urine is stained by bile. It is very doubtful whether the saliva, buccal, and alimentary secretions ever are; but it is said that tears and milk may be. It must, however, be excessively rare. I have never seen any of these secretions stained by bile, but inflammatory secretions usually are; thus if a jaundiced patient suffers from pneumonia, the expectoration is stained yellow.

The circulation of bile in the blood often gives rise to a bitter taste in the mouth.

When bile cannot reach the intestine the amount of gastric juice is increased, and there is therefore a considerable total hyperacidity. It is not known

whether the absence of bile from the intestine leads to any alteration in the pancreatic secretion or the succus entericus. The motions are a pale drab colour; they are commonly said to be "clay-coloured," and they contain a great excess of fat—it may be 70 per cent. The view commonly held is that the absence of bile from the intestine leads to constipation, but this is incorrect. Usually the motions are copious and loose, and this is what we should expect, considering the large amount of fat. The lack of colour in the fæces is due to the deficiency of urobilin. Often, no doubt, much of the excess of fat is owing to the fact that the same cause which prevents bile reaching the intestine also hinders the exit of the pancreatic juice with its fat-splitting ferment; for example, a large mass of malignant disease will do both. But apart from this, both clinical experience and experiment upon animals show that the mere absence of bile from the intestine leads to very fatty motions, for bile facilitates the absorption of all forms of fat. The absorption of proteids and carbo-hydrates is not interfered with by the absence of bile. The motions are soft and very bulky from the excess of fat; the proportion of water is not altered. Their specific gravity often falls below 1000, and the fatty matter floats on water; the free fatty acids cause the reaction to be strongly acid, and crystals of these acids may cause the motion to glitter. There are also present crystals

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of calcium salts of the fatty acids, chiefly palmitic and stearic, and perhaps minute amounts of the magnesium and sodium salts of the same acids. The proportion of fatty acids and soaps to neutral fat is the same as in health, namely 3 to 1. The failure to absorb so much fat leads to considerable loss of weight; it might be thought that this could be compensated for by giving more proteid and carbo-hydrate, but the hyperacidity of the gastric juice and the circulation of large quantities of bile in the blood cause so much indigestion that the quantity of food the patient will take is limited.

The pale putty-like stools of complete jaundice often have a disagreeable smell, and it is commonly stated that this is due to excessive proteid decomposition in the gut; this being especially active, because the antiseptic bile cannot reach the intestine. But there is no evidence that the bile is antiseptic; micro-organisms flourish in the gall bladder, and if in cases of obstructive jaundice there were excessive decomposition in the intestine, we should expect signs of enteritis, but we certainly do not meet with them. The disagreeable smell of the stools is due to the fatty acids.

The blood serum of a jaundiced patient is stained yellow. The coagulation time is prolonged; instead of being about four minutes, which is normal, it may be fifteen or twenty. This has an important bearing on the fact that patients with jaundice are very

liable to bleed ; thus surgeons often have difficulty in stopping bleeding when they operate upon jaundiced patients, and it is common to see hæmorrhages under the skin and mucous membranes of those who have had jaundice for some time. The pulse may be slowed, especially during the first few days of jaundice, a fact which harmonises with the suggestion that the slowing is due to the presence of bile salts in the circulation, for the liver probably soon ceases to form them. The blood pressure is usually low. Bile in the blood does not cause anæmia or any breaking up of the red cells, although when added to blood in a test tube they are broken up ; this is because there is never in a case of jaundice enough of the bile salts in the blood to disintegrate the red corpuscles. Bile in the blood has no effect on respiration or the temperature of the body, except that as death approaches both are depressed.

Nervous symptoms are often seen in association with jaundice. The most striking is that towards the end of a long-standing case of jaundice the patient gradually sinks into a drowsy state, in which he takes no notice of what is going on around him, and from which he cannot be roused. The respiration and pulse are hardly perceptible, the muscular weakness is extreme, and it is often difficult to tell whether the patient is alive or dead. To this condition the term cholæmia is applied. Occasionally such patients may suffer from convulsions or from

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delusions, but as far as my experience goes both are rare. These symptoms obviously cannot be due to the presence of bile salts in the blood, for after the first few days of jaundice these salts disappear; but they may be the cause of the intense mental depression, headache, and feeling of languor so very common in the early stages of jaundice. These early symptoms are often seen in catarrhal jaundice, and, if associated with persistent jaundice, they soon pass off.

Every student, when enumerating the symptoms associated with bile in the blood, mentions xanthopsia or yellow vision; but it is rare, and when present is usually slight. It has been thought to be due to the infiltration of the media of the eye with bile pigments, which it is suggested cause the absorption of blue light, and hence the patient sees things yellow. Certainly some jaundiced patients are blind for blue; but the weak part of the above explanation is that xanthopsia is often not present when jaundice is deep, and may be met with when it is slight.

At the post-mortem examination of a patient who has died jaundiced, the blood is found tinted with bile, and so are any pathological effusions, *e.g.* pleuritic or peritoneal. The clotted blood in the heart is also stained yellow. If the patient has pneumonia the solid lung has a yellow tint and exudes yellow froth, and the same is true of the

serum that exudes from any œdematous organ, *e.g.* the lung. If the post-mortem were made directly after death, probably none of the organs would be stained, but post-mortem imbibition takes place rapidly; hence most of the tissues are stained yellow. This staining is usually best seen in the aorta, but it may be seen almost anywhere and is often very evident in the liver and muscles. The brain and cord are very rarely stained, and then only faintly. The liver is, because of the damming back of the bile, uniformly enlarged if the obstruction to its outflow is considerable. The gall bladder is not enlarged in cases of toxic jaundice, but it is often considerably enlarged when there is gross mechanical obstruction of the common duct, *e.g.* malignant disease, unless such obstruction is a gall-stone, in which case the gall bladder is contracted. This is because the very presence of gall-stones shows that there has been some cholecystitis, and this leads to shrinking of the gall bladder. Pressure on the cystic duct leads to a large gall bladder full of clear mucus, and pressure on the hepatic ducts leads to no alteration in the gall bladder.

The Treatment of Jaundice.—Although this will to a large extent depend upon the cause of the jaundice, yet there are certain facts, especially as to diet, which must be borne in mind. Clinical experience teaches us that persons who have jaundice have a great repugnance for fats, which is what we

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should expect, for we have seen that when no bile reaches the intestine the absorption of fat is very considerably impaired. Hence, whatever the cause of the jaundice the patient should take no fat. If the cause of the jaundice is one that lasts a long while, it will be a good thing to inject oil under the skin; if this is done the oil is absorbed. I have injected sterilised olive oil under the skin of a patient who had an intestinal fistula high up in the jejunum, so that much of his food taken by the mouth passed out unused; it was impossible, owing to a kink, to inject food into the lower piece of the ileum. All the oil was absorbed, and the patient by dint of feeding this way, by the mouth and by the rectum, gained weight and strength until he reached a condition that it was possible by resection to restore the continuity of the gut. He made a complete recovery. The oil was injected under the skin of one thigh each morning and under the skin of the other each evening. Some authors have suggested an ounce of sterilised olive oil night and morning, but as the injection is painful half an ounce is probably all that can be borne for each injection. This is an ounce a day, or about 300 calories of food.

As the intake of fat by the mouth is so restricted, patients with jaundice waste, quite apart from the cause of the jaundice; the wasting is often in excess of that which can be attributed to the absence of

fat in the food. There are several reasons for this. The bile in the blood leads to dyspepsia; often the cause of the jaundice gives rise to dyspepsia, and perhaps the bile in the blood leads to an excessive proteid decomposition, but this is not certain. It is clear that the patient should take plenty of carbohydrates and proteids,—as much indeed as his limited powers of digestion will allow—and that his feeding should be little and often, especially as we have seen that he suffers from an excessive secretion of gastric juice. Many vegetables are either difficult of digestion or they contain so much water that their nutritive value is low; but lentil flour, which contains 22 per cent. of proteid, 65 per cent. of carbo-hydrate, and only 1·5 per cent. of fat, is a good food for those with jaundice. Potatoes properly cooked are desirable, for they contain about 20 per cent. of carbo-hydrates and very little fat. They should be steamed or cooked in their skins, for if not they lose a considerable part of their proteids and salts. Bread, toast, and biscuits are excellent, especially with jam or marmalade. If they do not cause indigestion, maltine and honey are valuable. Rice, tapioca, sago, arrowroot, grapes, and bananas may all be given. Most infants' foods contain very little fat, so they too may be used, and as whey and butter-milk are each of them milk from which the fat has been removed they are suitable to drink. The patient will get some proteid in each of the

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articles of food already mentioned, but he may in addition take fish, meat, chicken, or game. Inasmuch as the degree of indigestion varies much in different cases, it is difficult to give a dietary equally suitable for all jaundiced patients, but the attempt must be made to keep up the patient's weight by using foods judiciously chosen from among those just mentioned. Tea, coffee, and a moderate amount of alcohol may be taken.

REFERENCE

Herter. *Lectures on Chemical Pathology.*

DISEASES OF THE VESSELS OF THE LIVER

DISEASES OF THE PORTAL VEIN

By far the most important of these is thrombosis, and as the clotting is always associated with some disease of the wall of the vein, the condition is sometimes spoken of as portal thrombosis, sometimes as pylephlebitis. There are two varieties, suppurative and non-suppurative.

Non-suppurative Pylephlebitis.—The most common cause for this is cirrhosis of the liver, which accounts for between a third and a half of the cases; nevertheless it is a very rare complication of cirrhosis of the liver, being present in only 3 per cent. of those in whom cirrhosis of the liver is found after death. Long-lasting increase of the blood pressure in the portal vein is likely to lead to chronic inflammation of its wall, just as the pulmonary artery becomes thick and atheromatous in cases of mitral constriction; and it is very likely that this predisposes to clotting in the portal vein in cirrhosis. Further, the stagnation of the flow through the portal vein will contribute; but micro-organisms

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play a very important part in the formation of thrombi, and they are no doubt the determining cause of the thrombosis to which the thickening of the wall of the vein and the slow circulation through it predispose. The resisting power of patients with cirrhosis is low, so that micro-organisms easily get a foothold.

Malignant disease within the abdomen is the next most common cause for portal thrombosis; but although abdominal malignant disease is the cause of 10 per cent. of the cases of portal thrombosis, yet this is a very exceptional complication of malignant disease. Thus malignant disease of the stomach, liver, intestine, or pancreas may ulcerate its way into the lumen of some branch of the portal vein; this leads to some inflammation of the wall of the vein and to clotting within it. From the branch the process spreads to the portal vein itself.

Pressure upon a branch of the portal vein may lead to phlebitis and thrombosis, which spreads to the main trunk; such pressure may be exerted by many causes, *e.g.* a tumour, malignant or innocent, an aneurism, inflammatory adhesions in connection with a gastric or other ulcer, perihepatitis, chronic pancreatitis, a biliary calculus, or a gumma. Occasionally a blow on the abdomen has been followed by portal thrombosis. In a few cases thrombosis of the portal vein has been associated with thrombosis in other parts of the body, and it may occur to a

mild degree oftener than we think, for it is not uncommon to meet with patients—some of whom have had influenza, pneumonia, or typhoid fever—who suffer from successive attacks of thrombosis in various veins.

In rare instances some acute inflammatory lesion, such as an attack of appendicitis or cholangitis, leads to non-suppurative thrombosis, infection taking place at the periphery of the branches of the portal vein; in most of these cases the pylephlebitis is only non-suppurative because the patient has died before suppuration has occurred. In spite of the numerous causes for non-suppurative pylephlebitis here mentioned, there remain cases in which no explanation for the thrombosis can be discovered.

Non-suppurative pylephlebitis is more frequent in men than women, and usually occurs somewhere between the ages of thirty and fifty years, because cirrhosis of the liver is commoner in males of this age.

The first thing to be looked for at the post-mortem examination is the cause of the thrombosis. Next we examine the vein and find it hard and distended from the contained clot. If the inflammation of its wall has lasted a long while, as is often the case in cirrhosis of the liver, the wall will be tough and thick, and indeed in exceptional cases it is calcified like an atheromatous aorta. These chronic changes may be present without any thrombosis.

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If the cause of the thrombosis has not been fatal, the clot may have become completely organised to fibrous tissue firmly adherent to the wall of the vein; subsequent shrinking of this fibrous tissue leads to the conversion of the vein and its contained clot to a fibrous band. On opening the vein, if the thrombosis is recent, the wall is seen to be red and soft; the clot is seen in various stages of organisation, depending upon the time that has elapsed since it began to form. If recent, it shows the usual characters of ante-mortem clot. When, as is commonly the case, it is firmly adherent to the venous wall, the term adhesive pylephlebitis has been applied. Sometimes the clot does not fill the entire lumen of the vein; sometimes it softens and breaks down in the centre. If the lumen is completely blocked, the branches peripheral to the block are much dilated, and in a long-standing case the dilated veins of the collateral circulation are seen all around the blocked portal vein. There is the case on record in which symptoms of portal obstruction had lasted twenty years, and at the post-mortem examination the portal vein was a mere fibrous cord; in such a case the collateral circulation must have been very efficient.

No characteristic effect is produced upon the liver as a result of portal thrombosis. If the thrombosis is septic there may be abscesses in the liver (see portal pyæmia, p. 79). As might be expected, the

spleen is often enlarged considerably, and when the thrombosis is extensive there may be hæmorrhagic necrosis of the intestinal mucous membrane, usually of the middle of the jejunum.

Non-suppurative pylephlebitis is not often diagnosed during life, for the symptoms of it are almost always overshadowed by those of the cause. Analogy from the œdema of the leg that occurs with thrombosis of the femoral vein would lead us to expect that ascites would frequently occur, and as a matter of fact it is present in about 60 per cent. of the cases; but then ascites is a frequent symptom of both cirrhosis of the liver and malignant disease, the commonest causes of non-suppurative pylephlebitis. It used to be taught that very rapidly recurring ascites indicated portal thrombosis, but we know now that ascites recurs rapidly in other conditions, *e.g.* chronic peritonitis quite apart from portal thrombosis. Still, no doubt, it does strongly predispose to recurrent ascites. Although, as we have just seen, the spleen is often enlarged, yet the concurrent ascites or malignant disease often renders it difficult to feel, and it is enlarged in cirrhosis quite apart from portal thrombosis, which doubtless renders the patient especially liable to gastric and intestinal hæmorrhage, and tends to lead to dilatation of the veins around the umbilicus; but here again these symptoms may be due to cirrhosis quite apart from portal thrombosis, so they are of little value in diagnosing it. I find

students frequently forget that, with the exception of a few small veins around the umbilicus, none of the veins which dilate to accommodate the collateral circulation in portal obstruction are visible on the surface of the body. Indeed, non-suppurative pylephlebitis is almost impossible to disentangle at the bedside from its commonest causes. If suspected, it might be well to give the patient citric acid to diminish the coagulability of his blood.

Suppurative Pylephlebitis is a condition in which the portal vein contains pus and broken-up blood clot, resulting from the disintegration of a thrombus; often the same material is found in the branches of the portal vein in the liver, and sometimes in the peripheral branches of the vein; indeed, it may be present in any or all of these three situations. In a severe case the vein and most of its branches are full of thick greenish or brownish red foul-smelling pus. Suppurative pylephlebitis is so often caused by an infecting lesion in one of the organs drained by the portal system, that, when no lesion can be found, it may be assumed with confidence that it is so small that it has been overlooked. In 40 per cent. of the cases the lesion is an appendicitis; the next most frequent but much rarer cause is cholangitis due to a gall-stone; other causes are, ulcers of the stomach, duodenum, colon, or rectum. Sometimes the origin is in the female pelvic organs. It has been known to follow suppurating hæmorrhoids,

suppurating mesenteric glands, and an abscess of the liver or spleen; indeed, it may occur after any breach of surface at the periphery of the portal vein. The strange thing is that, considering the numerous diseases—*e.g.* typhoid and dysentery—which must implicate the minute veins which ultimately discharge into the portal vein, suppurative pylephlebitis is so seldom seen. That it is rare is shown by the fact that we only find one case in every thousand post-mortem examinations held at Guy's Hospital. It is clear that, as it is so rare and 40 per cent. of the cases follow appendicitis, the other causes must be extremely infrequent. As an instance in which no lesion at the periphery of the portal vein was found, I may mention a case under my own care in which it followed an empyæma; possibly some of the pus coughed up was swallowed, and the infection took place through some minute abrasion of the gastric or intestinal mucous membrane. It does not follow acute general peritonitis; no doubt because there is no damage to the minute branches of the origin of the portal vein. The micro-organism which is the cause of the trouble is often the bacillus coli; but others are streptococci, staphylococci, diplococci and perhaps the dysenteric amœba.

At the post-mortem examination, in addition to the primary focus, the broken-down blood clot and the pus in the vein and its branches, we find

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the walls of the portal vein and its branches are reddened and acutely inflamed; at first the clot is adherent to the walls of the vein, but, as softening of it progresses, it becomes detached. The suppurative process in the vein may be so acute that it extends through it, and an abscess forms outside it, but this is quite exceptional. If the patient dies very early, the liver may appear quite healthy, but it is nearly always found diseased, for the suppuration extends up the portal vein to the liver, and septic emboli are carried from the portal vein to the liver; as a result the liver is found to contain many abscesses and foci of necrosis. There may be hundreds of small abscesses containing foul pus; dissection of the branches of the portal vein shows that they are derived from it; indeed, these branches are often tubes of pus. The necrotic areas are of a dirty grey colour; sometimes hard, but more often soft and diffuent from mixture with pus. They may be any size, from being only just visible up to the size of half-a-crown; when seen in section they often have an arborescent arrangement suggestive of their origin in the minute branches of the portal vein. If adjacent abscesses or necrotic areas unite, their size may be considerable. Swarms of micro-organisms may be found in the pus and necrotic tissue. At first sight the condition might be confounded with suppurative cholangitis resulting in abscesses scattered through the liver, but a careful

examination of the bile ducts and portal vein will usually prevent a mistake. The liver itself is darkly congested and uniformly enlarged, the right lobe being affected more than the left. The abscesses and necrotic foci may be seen on the surface, often causing some local swelling and local perihepatitis. In many cases inflammation spreads to the bile ducts and a cholangitis results, but this is not suppurative.

The severe inflammation of the liver often spreads through the diaphragm, so that right-sided pleurisy is common. This may be followed by an empyæma, but apart from this the base of the right lung may show signs of congestion and pneumonia, with or without abscesses.

Peritonitis is found in 50 per cent. of the cases; nor is this surprising, for it is often due to the primary cause of the suppurative pylephlebitis, *e.g.* appendicitis, or it may be due to direct infection from the liver abscesses; some local peritonitis increasing the adhesion of the diaphragm to the liver is quite common. The spleen is usually enlarged and soft; occasionally the pancreas contains abscesses, because the pylephlebitis has spread into the pancreatic veins. Ascites is not a symptom.

If any of the hepatic abscesses or necrotic foci infect the hepatic veins, dissemination of the infective material through the general circulation will take

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place, but it is excessively uncommon for the hepatic veins to be implicated.

The disease is very rare, and is usually associated with a condition which itself gives rise to severe symptoms; therefore suppurative pylephlebitis is often overlooked. But nevertheless the interpretation of the symptoms is not difficult, for suppurative pylephlebitis is a variety of pyæmia in which the infective micro-organisms enter the blood at the periphery of the portal system, and produce local changes in the portal vein and liver. It is that variety of portal pyæmia (see p. 79) which is accompanied by thrombosis of the portal vein. This thrombosis may be very difficult to determine, but the diagnosis that the patient has portal pyæmia can often be made. As Dr. F. Taylor points out, we have the signs of the original local disease; secondly, the development of a definite pyæmic condition; and, thirdly, the evidence of hepatic disease.

The diagnosis of the original local disease does not concern us here; but it ought to be looked for most carefully, for the difficulty of diagnosis is enormously increased if we can find no source of infection at the periphery of the portal vein.

The development of definite pyæmia is recognised by the usual signs, namely, rigors, which occur in only half the cases, great oscillations of temperature, sweating, which is not so common as in other pyæmic conditions, great prostration, rapid feeble pulse, drowsi-

ness, anæmia, a sallow complexion, and the other well-known signs of pyæmia. In passing it is worthy of note that the severity of infection may be so great that the blood shows no leucocytosis; but it is often present, and the blood shows an extreme degree of secondary anæmia. The onset of this stage is often sudden.

The third group of symptoms occurs later. The liver becomes tender; the patient complains of pain over it, and occasionally a hepatic peritoneal rub may be heard; it is somewhat enlarged; the enlargement is uniform, and the organ may be felt one or two fingers' breadth below the ribs. As just pointed out, the right lobe is more affected than the left, and the right lobe may occasionally be so large as to cause slight bulging of the right lower ribs; and in one case under my care there were so many areas of necrosis and abscesses in the upper middle part of the right lobe that we made out a dome-shaped increase of the dulness upwards in the mid-axillary line, and putting in a needle obtained pus. But this is quite unusual; often the enlargement of the liver can hardly be detected clinically. Rather less than half the patients are jaundiced, because the inflammation in the liver spreads to and blocks the smaller ducts. The jaundice is usually bright yellow and not very deep. When it is present the urine contains bile; but, inasmuch as there is not total obstruction to the exit of bile, the fæces

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are usually coloured, but may be pale. Diarrhoea is often present; sometimes it is sanguineous. This is not surprising, considering that the portal vein is blocked. The spleen may be felt to be enlarged. If the inflammatory process spreads through the right wing of the diaphragm we may find signs of right-sided pleurisy. The patient is usually so ill that we can hardly detect the general peritonitis that is so often associated with suppurative pylephlebitis. Albuminuria and vomiting are common. The patient becomes weaker and weaker, and dies comatose.

That the patient is suffering from some form of pyæmia can often be made out if care is taken; and if the symptoms due to the originating lesion in the periphery of the portal vein lead us correctly to it, or we observe evidence that the liver is affected, we can diagnose portal pyæmia, although not even under the most favourable circumstances can we be sure whether there is suppuration in the portal vein, for in portal pyæmia the suppuration is often confined to the liver. The cases in which we have no clue to either the lesion in the periphery of the portal vein, or to the disease of the liver are very difficult, and are usually mistaken for general pyæmia, typhoid fever, or malignant endocarditis. We have seen that the right pleural cavity and the base of the right lung are often affected; hence, suppurative pylephlebitis has been thought to be pneumonia, empyæma, and subdiaphragmatic abscess.

The outlook is very grave ; only one or two cases of apparent recovery are known. No treatment appears to do good ; possibly vaccine or serum treatment may in the future hold out some hope.

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CONGESTION OF THE LIVER

CONGESTION of the liver is a term commonly used by the public to describe various symptoms of indigestion, but there is no evidence that such symptoms are due to disorder of the liver. We only certainly know of two varieties of congestion of the liver, that which follows backward pressure from disease of the heart or lungs, and that which is met with in the tropics and is usually called tropical liver.

VENOUS CONGESTION FROM BACKWARD PRESSURE

When the liver is thus congested a section of it looks like a section of a nutmeg. By far the most frequent cause of this congestion is mitral disease; the most potent cause is tricuspid stenosis, but this is very rare. It may follow upon pericardial adhesions, which hamper the action of the heart, and occasionally it results from imperfect action of the heart, owing to disease of the cardiac muscle. Nearly all the instances of nutmeg liver seen in the post-mortem room are caused by heart disease, but in a few the damming back of venous blood in the liver is not due to impaired action of the heart but to obstruction of the venous flow through the lungs.

By far the most frequent pulmonary condition leading to this result is chronic bronchitis, especially when associated with emphysema. It is a remarkable fact, for which no satisfactory explanation is forthcoming, that nutmeg liver is not seen in cases in which large areas of lung are destroyed or converted into fibrous tissue by phthisis. We certainly might have expected that nutmeg liver would follow, for many branches of the pulmonary artery must be obstructed. As mere curiosities it may be mentioned that tumours pressing upon the inferior vena cava and obliteration of it have led to a nutmeg liver.

When we look at a nutmeg liver we are struck by its increased size, by the fact that it is of a darker red than normal, by the fact that the enlargement is perfectly uniform, and often by patches of local perihepatitis, which may be seen here and there on it; indeed, increased backward venous pressure is the most frequent cause of local chronic perihepatitis. If the heart is enlarged there may be an increase of the natural slight depression produced upon the liver by the heart. When the liver is cut across, it is seen to contain more blood than usual, and the distension of the intralobular and sublobular veins makes them unusually dark, large, and evident. The hepatic tissue of the lobule around them is of a paler yellow; partly because the liver cells have become fatty, partly because in some cases they are stained by bile. These pale lobules with the dark

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intralobular veins in their centre make the liver look very like a section across a nutmeg. The hepatic veins, too, are somewhat dilated, and the pressure exerted by all these dilated veins leads to some atrophy of the hepatic cells. It has been stated by more than one author that long-standing venous congestion of the liver leads to cirrhosis of it. No doubt, long-standing congestion of almost any organ leads to hardening of it—indeed, a nutmeg liver is somewhat hard—and under the microscope a trifling increase of fibrous tissue may be seen, as, for example, in the india-rubber kidneys and indurated lungs met with in cases of mitral disease which have lasted a long while; but however severe the backward venous pressure has been, we never meet with a nutmeg liver in which the increased fibrosis has been nearly enough to make the organ even remotely resemble the ordinary cirrhotic liver, unless some cause—usually alcohol—which commonly leads to cirrhosis has been present as well as the increased venous pressure. A nutmeg liver is often smaller after death than during life, for when it is taken out of the body some blood drains out of it.

The symptoms produced by a nutmeg liver are such as might be expected. The patient complains of a feeling of tightness, discomfort, and heaviness in the hepatic region, and unless the abdomen is distended or the chest emphysematous, the liver can be felt to be firm and enlarged; sometimes it may extend

four or five fingers' breadths below the ribs, the enlargement is uniform and smooth, the patient often complains of considerable pain, and the organ is tender. The pain and tenderness are no doubt due largely to the distension of the hepatic capsule, for it must be remembered that the liver can contain a great deal more blood than is natural to it; sometimes, however, the pain and tenderness are caused by the patches of perithepatitis. The liver is rarely enlarged upwards; at any rate, percussion cannot detect any upward enlargement, so if present it must be very slight. This is because it will enlarge in the direction of least resistance, namely, downwards; and further, the greater quantity of blood in it adds to its weight, so that it will tend to drop down, especially as the subjects of long-standing mitral disease often have weak abdominal muscles. The skin over the enlarged liver is often tender, but then we know from Head's researches that when viscera are diseased certain areas of the skin, which get their nerve supply from the same areas of the cord as the diseased viscera, are tender.

Patients with a severe degree of nutmeg liver may be slightly jaundiced; probably this is due to pressure exerted by the dilated veins upon the minute bile ducts. When jaundice appears in the course of heart disease the outlook is grave.

Many symptoms referable to the gastro-intestinal tract, such as vomiting, constipation, flatulence,

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pain after food, &c., are often present, but there is no evidence that they are due to venous congestion of the liver; they may be caused by the general difficulty of the return of the portal blood from the stomach and bowels, for the rise of venous pressure in the hepatic veins must retard the portal flow. In 235 cases of heart disease of sufficient severity for the tricuspid orifice to be incompetent, Newton Pitt found œdema of the lower extremities in 200 and ascites in 140, œdema and ascites occurred together in 124 cases, œdema alone in 76, and ascites alone in only 14, and these figures suggest that the view commonly held, that the ascites of heart disease is usually only part of the general œdema, is correct. Ascites is not of the same serious significance in these cases as it is in cirrhosis. It need hardly be added that patients with nutmeg liver have, in addition to the symptoms of this, those of heart or lung disease as the case may be, and those of venous congestion of other organs. For the treatment of nutmeg liver, see p. 66.

Pulsation of the Liver.—When the tricuspid orifice is incompetent and the right ventricle is beating sufficiently forcibly, a pulse wave travels back in the inferior vena cava and hepatic veins to reach the liver and make the whole organ expand synchronously with each beat of the right ventricle. In such cases the tricuspid regurgitation is nearly always secondary to mitral disease; but

inasmuch as whether or not the liver shall pulsate depends upon the contractile power of the right ventricle, we sometimes meet with hepatic pulsation in cases in which the mitral disease is not extreme. On the other hand, we may find it absent when the mitral disease is very advanced. Great care must be taken not to mistake a thrust downwards of the liver by the contraction of a hypertrophied heart, or the thrust forwards of the liver by pulsation of the aorta for hepatic pulsation. The distinguishing feature of this is that, when one hand is placed on the front and the other on the back of the abdomen over the enlarged congested liver, the two hands can be felt to be separated by the expansile pulsation of the liver; this is not the case when pulsation is simply transmitted to the liver from the heart or aorta. It is quite common to find pulsation of the jugular veins associated with pulsation of the liver. It follows from what has been said that pulsation of the liver is synchronous with contraction of the right ventricle; but in those very rare cases in which tricuspid stenosis is present the pulsation, not only of the liver but also of the jugular veins, will naturally be synchronous with the contraction of the right auricle. Pulsation of the liver that can be felt by the hands is rare; thus out of 235 cases of tricuspid regurgitation analysed by Dr. Newton Pitt it was present in 15 only, and in only 8 out of 87 cases of tricuspid

stenosis; but Mackenzie, by taking tracings of the liver with the polygraph, showed that some pulsation of the liver was often present when it could not be detected by the hands. It is stated that when pulsation of the liver has once appeared it persists until the end of the case. Even if this is true of those cases in which, by means of the polygraph, care is taken to detect slight pulsation, there is no doubt that under treatment and rest in bed pulsation of the liver tangible to the hands may diminish and pass away.

The treatment of venous congestion of the liver is principally the treatment of the cardiac or pulmonary disease which gives rise to it; but it may be frequently observed that the local pain so often associated with a large nutmeg liver can be much relieved by the application of several leeches to the abdominal wall over the liver, and by the administration of calomel at night, followed by a saline aperient in the morning.

General Congestion of the Liver.—Many persons when they over-eat and take insufficient exercise feel out of health. They complain of lassitude, slight headache, loss of appetite and irregularity or constipation of the bowels; their urine is scanty, high-coloured, and often contains obvious lithates. There may be a feeling of fulness at the upper part of the abdomen. When it is not clear to what these symptoms are due, some ascribe them to congestion

of the liver ; but we have no post-mortem or direct evidence that the liver is congested. If the patients can afford to go they are usually sent to Marienbad, Carlsbad, or Harrogate. Certainly they often come back much better. This is probably due partly to care in feeding and partly to the purgation which follows taking natural saline aperients. If they cannot go to any of these places, restriction of diet, regular exercise, and the taking of a little calomel at night, followed by a saline aperient in the morning, will generally lead to improvement. A usual prescription is a pill containing a grain or half a grain of calomel, with the same quantity of dried extract of euonymin, to be taken directly after dinner, and followed next morning by half a claret glass of *Æsculap* water, with an equal quantity of warm water. This may be sipped during dressing. If there is much pain in the upper part of the abdomen, and especially if it is over the liver, a hot pack or the local application of leeches often gives relief. It is only fair to add that many consider that congestion of the liver exists in these patients because the liver, they state, is enlarged and tender ; but in this country this is certainly exceptional. If the liver is enlarged and tender it returns to its natural size, and the tenderness passes away if the above treatment is adopted. The precise dietary ordered probably matters but little, so long as the food is plain, easily digested, not too

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abundant, and eaten slowly. Nevertheless, it is often desirable to write out a dietary, for some patients are gluttons and will not submit to a restriction in their diet unless what they are to eat is written down in black and white.

Tropical Liver.—If a European lives for many years in the tropics he is very liable to suffer from attacks of congestion of the liver, and as there are many English residents in India the disease is often known among Englishmen as Indian fever. One attack of congestion succeeds another, until at last the liver is chronically congested; but even then the patient often suffers from attacks of acute congestion in addition. The liver becomes uniformly enlarged, it is smooth, somewhat harder than is natural, and it bleeds readily on section. It is said that in long-standing cases there is some increase of fibrous tissue, but unless, as is often the case, the patient has taken more alcohol than he should, any increase of fibrous tissue is trifling, and does not lead to such a condition of the liver that it could be mistaken for an ordinary cirrhosis.

Those physicians whose residence in the East makes them familiar with tropical liver, consider that the most important causes of the condition are great heat and errors in diet. Certainly they are supported in this view by the fact that lately the trouble has become much less frequent, and it is of late years that European residents in India have

been taking leave from time to time, during which they have returned to England for some months, and also they have, as a class, been more careful in their diet, especially they have taken less whisky. It is notorious that different individuals are differently affected by heat; therefore some become the subjects of tropical liver more readily than others, but it appears clear that sudden variations of temperature are particularly harmful; hence the origin of the phrase "a chill on the liver." Therefore when a European has been exposed for some time to considerable heat, and is consequently sweating profusely, he should not expose himself to a draught and so lower his temperature rapidly, nor should he take a cold bath. Europeans who have resided a long while in India frequently get out of health on returning to England, where they are liable to exposure to greater cold than was ever the case in India. Attacks of congestion of the liver also follow upon great fatigue.

Plain, simple food, with very little alcohol and not much of highly-spiced articles of food, such as curry, which easily cause congestion of the stomach, is important for those living in India. Failure to remember this increases the liability of hepatic congestion. Rational exercise, short of fatigue and taken in the cool of the evening, helps to ward it off, but no amount of exercise will prevent it if much food, especially animal food, spices, and

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alcohol, is taken. It is a true saying that each man has the liver he deserves. Repeated attacks of malaria and dysentery are believed to predispose to hepatic congestion, but whether they do so directly and whether merely as a result of lowering the general health is not known.

The symptoms of tropical liver are at first want of appetite, a furred tongue, a bitter taste in the mouth, heartburn, indigestion, flatulence, headache, and a general feeling of languor. The sufferer complains of a sense of oppression in the hepatic region; he may have actual pain, and after a time the liver becomes uniformly enlarged and tender on pressure. The bowels are constipated and the patient is depressed. If the attack is more acute, there may be a rise of temperature to two or three degrees above normal, the pulse is increased in frequency, the liver is acutely painful and tender; the pain may radiate widely over the right side, even up to the shoulder. The motions are pale, and the constipation may be from time to time replaced by diarrhoea. The urine is high-coloured and contains an excess of urates. When the condition is acute, it is often spoken of as hepatitis, and I have known the sufferer from it to be erroneously thought to have an abscess of his liver.

If the disease is chronic, the liver is uniformly enlarged—it may reach to three or even four inches below the ribs—there is a constant sense of weight

over it, the appetite is poor, digestion slow, the tongue remains furred, the patient complains of chronic indigestion, constipation is troublesome, the urine contains an abundance of urates, the complexion is sallow, and there is a faint tinge of yellow about the conjunctivæ, although no actual jaundice of the skin. The patient becomes depressed, morose, and irritable. If he has lived too freely the symptoms of dyspepsia are very striking, and if he has had malaria the spleen is enlarged.

Treatment.—By being careful in his diet, by avoiding alcohol, by regular active exercise in the cool, by avoidance of exposure to great heat or fatigue, and by suitable clothing, Europeans can do much to prevent suffering from hepatic congestion. When the attack is mild, all that is necessary is for the patient to take at night a pill containing a little calomel, with either podophyllin or euonymin, and follow it up by a saline aperient before breakfast. This may be continued for a few days according to the severity of the case. The diet should be restricted to plain, simple food, and a mixture containing 10 minims of tincture of nuxvomica, 10 minims of dilute hydrochloric acid with a drachm of tincture of gentian in an ounce of chloroform water may be given three times a day. If the attack is acute the patient should be put to bed, have a milk diet, and leeches or hot fomentations should be applied over the liver. If the patient

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is constipated, the same pill as that just mentioned should be given at night in such a dose that when followed by a saline draught in the morning the bowels will be thoroughly opened. If diarrhoea is present, it should not be checked, but powdered ipecacuanha root, in doses of 20 or 30 grains twice a day, should be given for two or three days. When the condition is chronic, the bowels should be kept open in the way just mentioned, and those who have much experience think highly of local packs of nitro-hydrochloric acid over the liver. Eight ounces of dilute nitro-hydrochloric acid are added to a gallon of water at a temperature of 98° F.; cloths soaked in the mixture are applied to the region of the liver and the upper part of the abdomen, and changed frequently. Spongiopiline may be used instead of cloths. If the trouble has lasted a long while, a holiday in temperate climate will do much good provided that great care is taken to avoid a chill. Taking the waters at Carlsbad, Marienbad, or Harrogate is often beneficial.

DISEASES OF THE HEPATIC ARTERY

THESE are excessively rare ; even the commonest, namely, aneurism, is quite exceptional.

Aneurism of the Hepatic Artery is probably most often due to septic embolism. This was the cause of the only case that I have had under my care. The patient, a medical student, had pneumonia, which ended by lysis on the ninth day of his illness. The temperature remained normal until the fourteenth day, when it became hectic, and remained raised till death. On the twentieth day jaundice was first noticed. On the twenty-fourth a localised empyæma under the right nipple was found and opened. A slight fulness was noticed over the liver. A month after his illness began he fell back dead while the nurse was washing his face. At the autopsy it was found that he had malignant endocarditis, probably pneumococcal, with an aneurism of the right branch of the hepatic artery about the size of a Tangerine orange. This had ruptured into the abdominal cavity, which contained much blood clot, and its sudden rupture was the cause of death. It pressed on the common bile duct and the portal vein.

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There was another aneurism springing from the left branch of the hepatic artery about half an inch in the liver substance. Both aneurisms contained ante-mortem clot.

Other instances are on record of an aneurism on both hepatic arteries, suggesting that an embolus impinging at the bifurcation becomes broken into two pieces. That septic embolism is the commonest cause of hepatic aneurism is supported by the fact, that out of twenty-four cases of hepatic aneurism collected by Rolleston one-third occurred in women; a much higher proportion of women than is found in the case of other aneurisms. Occasionally, no doubt, hepatic aneurisms are due to atheroma; in one case a hepatic aneurism appeared to follow a kick in the abdomen by a horse, and in one case an aneurism was found projecting into an abscess of the liver, the outer wall of the vessel having been weakened by inflammation due to the abscess, the mode of production being the same as that of a pulmonary aneurism in a phthisical cavity. An aneurism may be produced by the ulceration of the outer wall of the vessel from a gall-stone; perhaps the frequency of gall-stones in women has a little to do with the proportionately large number of hepatic aneurisms in them.

The disease is rarely diagnosed. When it is due to infarction the illness of the patient overshadows the symptoms of aneurism. In the case of the student it was recognised that he was septic; and as one

empyæma had been discovered, it was thought that the fulness over the liver and the pain there were due to the presence of a deep-seated empyæma under the base of the right lung; indeed, one was found there post-mortem, and the jaundice was regarded as caused by the intensity of the septic poisoning. As a hepatic aneurism is under the liver a pulsating tumour can hardly ever be felt; and even if such a tumour could be detected, the aneurism would probably be thought to be of the celiac axis, as that is the commonest seat of an abdominal aneurism. Jaundice is a common association of hepatic aneurism, but it may be seen in association with one of the celiac axis, and pain and a murmur may be present in both. Most of the cases which have not been septic, and in which neither tumour nor murmur could be discovered, have been regarded as examples of gall-stones if the patient has been jaundiced, or of duodenal ulcer if he has not.

Hepatic aneurism usually ruptures into the peritoneal cavity and so causes sudden death; less often it bleeds into the duodenum or stomach; then, if the patient live long enough, hæmatemesis or melæna may be seen.

If the condition is diagnosed and the patient is not suffering from septicæmia, the abdomen should be opened, for in one case recorded by Kehr the aneurism was dealt with successfully by surgery.

Embolism, thrombosis, and atheroma of the

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hepatic artery are very uncommon and of no clinical interest, except so far as embolism or atheroma may lead to aneurism.

Infarcts are very rare in the liver, but occasionally both anæmic and hæmorrhagic infarcts may be found in association with thrombosis or embolism of the portal vein, or with obstruction of the portal and hepatic veins or their branches by the pressure of a new growth, or with combined portal and hepatic vein thrombosis or with embolism of the hepatic artery. The very rarity of such infarcts shows that there must be some special reason why the liver should escape, and it is usually supposed that the reason is that the liver has a double blood supply for the capillaries of the hepatic artery anastomose with those of the portal vein. Why, indeed, infarcts should ever occur as a result of thrombosis of the portal vein is difficult to understand, in such cases there is probably some unknown and peculiar change in the blood which is the cause of the hepatic infarction, for Wooldridge showed that infarction of the liver could be induced by the injection of tissue fibrinogen into the jugular vein of a dog. Infarcts of the liver need not be further discussed, as they are of no clinical interest.

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SUPPURATION WITHIN THE LIVER

Multiple Abscesses.—Fortunately these are not common, and are becoming less frequent. There are many causes for them. The pus-producing micro-organisms may be conveyed to the liver by the bile passages, as in the very rare cases in which a gall-stone leads to suppuration in the bile ducts; in such a case the bile ducts are at the post-mortem examination seen as tubes of pus running through the liver. Secondly, as is usually the case, the micro-organisms may be conveyed by the portal vein; the case is then one of portal pyæmia. The source of infection is at the periphery of the portal vein; the micro-organisms are conveyed from there to the liver, where they set up a number of small abscesses at the termination of the radicles of the portal vein; often at the same time there are patches of necrotic liver tissue, and the intrahepatic radicles of the portal vein are thrombosed. Thirdly, the micro-organisms may be conveyed to the liver by the hepatic artery. We then have multiple pyæmic abscesses in the liver as part of a general pyæmic infection. In all the above instances of suppuration within the liver the abscesses are small and multiple.

Multiple abscesses of the liver form a distinct

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clinical group, both in the appearances seen post-mortem, in the symptoms, and in the unfortunate fact that they are invariably fatal. The variety in which the pus is in the hepatic ducts is quite uncommon, and belongs to Mr. Bland Sutton's book in this series on gall-stones, and need not therefore be considered here. The variety in which the pus-producing micro-organisms are conveyed by the hepatic artery—in other words, the hepatic abscesses as part of a general pyæmia—is extremely uncommon. Indeed, hepatic suppuration is so rarely associated with general pyæmia or malignant endocarditis that there must be some special reason within the liver why it should be so uncommon; even when abscesses are seen, they are quite small, and have during life caused no symptoms. The rarity of the affection of the liver is not solely due to the rarity nowadays of pyæmia, for when it was a common disease hepatic suppuration, as a part of a general pyæmic, was not often seen. Strange to say, although hepatic abscesses due to general pyæmia are so infrequent, they may be present in the liver when none are to be found elsewhere in the body; and it was long ago pointed out by Percival Pott that this was especially likely to be the case when the source of the infection was in the head. But as, at the present time, middle ear disease, the principal source of pyæmic infection in the head, is rarely allowed to progress to such a stage as to

give rise to general infection, the subject is of little practical interest.

The multiple abscesses in the liver that are of greatest clinical importance are those formed by micro-organisms arriving by the portal vein. The patient suffering from them is usually said to be suffering from portal pyæmia. The intensity of the infection may be such that he may die before many abscesses have formed, or indeed before any have formed, although areas of necrotic liver tissue and thrombosis of the intrahepatic radicles of the portal vein show that portal pyæmia was present. Still, although in very rare cases only one or two abscesses are present, yet it is remarkable that almost always, if any are to be seen, they are numerous. Sometimes, too, pyæmic abscesses form in the liver, the infection being conveyed by the portal vein, without any thrombosis of the intrahepatic branches of this vein being seen. This is particularly likely to be the case when the source of infection is dysenteric inflammation of the intestine. Sometimes pyæmic infection takes place from a solitary large abscess in the liver, so that post-mortem we find the large abscess with a number of small ones. It is strange that gastric ulcer and typhoid fever so rarely give rise to portal pyæmia; but it has been known to occur as secondary to both these conditions. Multiple pyæmic abscesses are usually near the surface of the liver, and of course more numerous

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in the right lobe than the left. Commonly they are about the size of a pea, but may be larger or smaller, and they are usually arranged in an arborescent manner, indicating that they have been formed in the course of the intrahepatic branches of the portal vein. If the patient lives long enough, which is very rarely the case, several may break down to form one rather large abscess; but usually he dies before all the abscesses are fully formed, and we see soft areas of dark necrotic liver tissue, with much hæmorrhage in them, and each area would no doubt soon have become an abscess. The liver itself is usually very dark in colour, and the pus often smells horribly.

Hepatic abscesses due to infection by the portal vein and suppurative pylephlebitis are really different varieties of the same disease, and therefore the description of the causes and symptoms of multiple hepatic abscess have already been given when describing suppurative pylephlebitis (see p. 52).

Large Single Abscesses in the Liver.—The classification of abscesses of the liver into small multiple abscesses and large single or solitary abscesses is of considerable practical importance, for most cases of hepatic suppuration can be put into one of these two groups which differ in their etiology and outlook, but just as the patient may—very rarely, as we have seen—die before pyæmic abscesses have had time to become numerous, so he

may die before an abscess which would have become large has had time to do so, and occasionally he lives long enough for two or even perhaps three large abscesses to form in his liver; yet, nevertheless, he belongs to the clinical group in which the hepatic abscess is nearly always solitary.

By far the commonest cause of the large single abscess of the liver is dysentery; the abscess which follows this is usually called tropical, because, although not confined exclusively to the tropics, it is more common there than elsewhere. Suppuration in a hydatid cyst and injury are less frequent causes. Very rarely indeed various specific fevers have been known to be followed by a solitary abscess of the liver, *e.g.* scarlet fever, typhoid, influenza, and even pyæmia. A single large abscess may result from suppuration about a gall-stone or spread of inflammation from some abscess in the neighbourhood, *e.g.* a perinephritic abscess. The large abscesses due to suppuration of a hydatid, to injury, to specific fevers (other than dysentery), to gall-stones, to spread of inflammation from the neighbourhood, are very uncommon, their cause is fairly obvious, and the symptoms to which they give rise are the same as those of "tropical abscess"; therefore, they will not be described separately.

Much has been written about the etiology of the large solitary tropical abscess of the liver, and it appears quite certain that it hardly ever follows upon

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the disease known as bacillary dysentery, which is quite distinct from the dysentery in which an amœba is found in the stools. For example, single hepatic abscess was excessively rare during the South African War, although bacillary dysentery was very common. Whatever may be the exact micro-organism which causes the intestinal ulceration met with in large institutions, especially asylums, and which is often seen in temperate climates, it certainly is not an amœba, and a large abscess of the liver does not result from this disease. Large abscesses of the liver did not follow upon the dysentery that broke out among the soldiers during the Franco-German War; the dysentery that occurs in the jails in India is bacillary and is not followed by this variety of abscess. When hepatic suppuration occurs in association with dysentery that is not amœbic, it consists in a number of small abscesses in the liver; it is really a portal pyæmia. In passing it is to be noticed that this is of great practical importance, for if hepatic suppuration occurs in the course of amœbic dysentery, as the abscess is single it may be successfully drained, or the patient may recover after it bursts into the lung or the colon; but, as we have already seen, recovery after pyæmic abscesses in the liver is virtually unknown.

As it is only of late years that we have learnt to draw a proper distinction between amœbic and bacillary dysentery, large numbers of figures are

not yet available. But, as far as they go, it appears that an abscess of the liver is found in about 23 per cent. of the fatal cases of amœbic dysentery; but as patients with amœbic dysentery usually recover, the number of cases of amœbic dysentery complicated by hepatic abscess is far less; it has been said to be about 6 per cent. It is certainly more frequent among Europeans living in the tropics than among natives. Hepatic abscess has been produced experimentally by feeding cats on amœbic stools; the abscesses were as a rule single but sometimes multiple. The statistics from different parts of the tropics vary somewhat in the proportion of cases of tropical abscess in which distinct evidence of dysentery can be obtained, but in most it can very often be ascertained. Thus Major Rogers, whose figures were taken from Calcutta, found both clinical and post-mortem evidence of dysentery in 55·5 per cent. of the cases of large hepatic abscess, post-mortem evidence only in 20·63 per cent., and clinical evidence only in 14·3 per cent., so that in only 9·57 per cent. of the cases was there no evidence of dysentery. Inasmuch as by the time the patient dies of his hepatic abscess, dysenteric lesions may have completely healed, and sometimes he may, when questioned, forget that he has had symptoms which indicate a mild attack of dysentery, these figures suggest that almost all cases of large hepatic abscess are due to amœbic dysentery, but

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not all authors have found such a large proportion of dysentery among those who die of hepatic abscess, and I shall be able to show that although solitary abscesses with intestinal ulceration may occur in temperate climates, yet here also we may find them without any intestinal ulceration or obvious cause; these rare cases in temperate climates are usually called "idiopathic" hepatic abscess, and in them the pus is usually sterile and contains no amœbæ. As the association between amœbic dysentery and hepatic abscess is so close, we find hepatic abscess almost confined to these places in which amœbic dysentery is found. Thus hepatic abscess is common in India, it is rare in Ceylon and the Malay Peninsula, it is common in the Philippine Islands, Java, and Sumatra, but rare in China; it is common in Egypt and Mauritius, moderately common in the Southern States of America, but very rare in the West Indies. It is curious that wherever it exists it is commoner in Europeans than natives, for the natives are as liable to amœbic dysentery. It cannot be too strongly insisted that if a person, who has previously lived in a part of the world where he is likely to have had amœbic dysentery, shows, even after some long residence in a temperate climate, obscure febrile symptoms difficult of explanation, they may be due to a solitary tropical hepatic abscess.

We now come back to the interesting subject of large abscesses of the liver in those who have not

had amœbic dysentery. Some of these are, no doubt, due to a blow which, however, is more likely to cause a subdiaphragmatic than a hepatic abscess; some are really not abscesses of the liver, but abscesses of the gall bladder which have invaded the liver; some are due to the spreading into the liver of abscesses due to disease of other organs; for instance, an appendicular abscess may extend into the liver; some are suppurating hydatids; some are examples of suppuration round a gall-stone impacted in the liver, and even an ascaris has been found in the centre of an abscess of the liver; some may be due to actinomycosis, some are rather large pyæmic abscesses; but it is very exceptional to find these large, probably they are hardly ever larger than a walnut, and even that size is exceptional; some examples of a large abscess in the liver appear to follow a specific fever, as has already been mentioned, but this is very uncommon. Every one of these varieties of large hepatic abscess is very rare, and we from time to time meet with cases in which the patient has never been out of Great Britain, yet he has a large abscess in his liver which post-mortem examination shows could not be explained by ascribing it to any of the causes just mentioned. It is extremely interesting to observe that a few of these large abscesses in those who have never been abroad, are associated with the disease known as ulcerative colitis. Some writers believe this to be a form of bacillary dysentery, others think it a

distinct disease; but, whichever view is correct, the association of it with hepatic abscess is equally obscure, for we have seen that in the tropics it is not the bacillary but the amœbic dysentery which claims hepatic abscess as its complication. The whole subject is very difficult to understand, for why should it be that, if the infection of the portal area takes place by the micro-organisms of, say, a case of appendicitis, the liver abscesses are small and multiple? and yet if the infection conveyed from the portal area is an amœba, the abscess in the liver is large and single? or, again, why is it that portal pyæmia is so rare although the intestine swarms with dangerous micro-organisms? The difficulty of understanding why an amœbic abscess should be single is the same as that of why a hydatid cyst should be single, as it usually is; we should have expected amœbæ or the scolices to be showered about the liver indiscriminately.

As an illustration of the association of a large single hepatic abscess with ulcerative colitis, I may take the case of a middle-aged man who had never been out of England, and said he had never had malaria or dysentery. He was admitted under my care with what was obviously a large abscess of the right lobe of his liver. He was extremely ill, and sank five days after the abscess was opened. It contained anchovy paste-like pus, which to the naked eye was exactly like the pus so often seen in an amœbic tropical abscess. At the post-mortem the whole of

the man's large intestine was riddled with ulcers, and was an example of what is commonly called ulcerative colitis. The abscess was very large, occupying a considerable part of the right lobe. There were several small pyæmic abscesses in the liver ; whether the infection causing them had come from the large abscess or the intestinal ulceration it is difficult to say. Other examples of a large hepatic abscess in association with ulcerative colitis in patients who have never been out of England have been recorded. For example, Fagge records two and Dickinson records two. In one of Fagge's cases there were two large abscesses in the liver.

Thus it appears undoubted that very rarely large solitary abscess of the liver may occur as an association of ulcerative colitis. As in some of the older cases, in which no operation was performed, the abscess burst into the bowel, the suggestion has been made that the intestinal ulceration was due to this ; but intestinal ulceration, dysenteric or otherwise, is so often associated with hepatic abscesses which have not burst into the bowel that this explanation will not hold. But even when we have excluded large solitary abscesses of the liver, due to injury and other causes mentioned on p. 85, and those associated with ulcerative colitis, there still remain some not due to amœbic dysentery. We are quite in the dark as to the cause of these, and so they are often called idiopathic.

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This name is only used provisionally, until we can find a cause for the abscesses. Some of them occur in temperate climates. Thus Fagge says: "Within the last twenty years there have been in Guy's Hospital 15 cases in which death was caused by the formation of a single large abscess in the liver. Five of these cases occurred in persons who had come from China or India or the West Coast of Africa, but in 10 of them there is no such history, and in the majority of them it is positively stated that the patients had never been out of England." I certainly have seen cases in persons who have never been out of England, and in whom neither ulcerative colitis nor any cause previously mentioned would explain the hepatic suppuration. But, as mentioned on p. 83, it seems clear that a few of the cases in the tropics are not due to amœbic dysentery. Many writers have recorded examples which prove this. Davidson concludes that some of the best observers have failed to find dysenteric or amœbic origin in from 15 to 30 per cent. of their cases. From the context it appears that when he uses the word dysenteric he is speaking of amœbic dysentery, but it must be remembered that occasionally bacillary dysentery leads to a large abscess in the liver; it has been suggested that this is sometimes due to the breaking into one another of several small abscesses.

To sum up, we may say that most of the large abscesses seen in the world are associated with

amoebic dysentery. A few are not so associated; of these a very few have some obvious cause or association, *e.g.* injury; others have no obvious cause or association. Provisionally these are called idiopathic hepatic abscesses. Even idiopathic abscesses are commoner in the tropics than in temperate climates, which seems to show that the amoeba is only at best a contributory cause. Certainly the European living in the tropics is much more liable to the single large hepatic abscess than the native; and the European who eats too much, and especially who drinks too much alcohol, is more likely than others to become affected. The depression in general health caused by malaria seems to be a predisposing cause. Some, indeed, think that malaria predisposes to hepatic abscess by a direct action on the liver. Men are much more often affected than women; how far this is due to the fact that men in the tropics as a rule drink more alcohol than women, that they are more exposed and work harder, is not known. Perhaps these two factors are not sufficient to explain the great preponderance of men, nor do we know indeed how great this preponderance is, for there are no exact figures showing how many more European men than women are resident in the tropics. Single hepatic abscess may occur at any age, even in childhood, but naturally it is mostly seen in adults aged between twenty-five and forty-five years, for younger Euro-

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peans do not often go to the tropics, and they usually leave not long after forty-five and fifty. Each year that a European lives in India increases his chance of getting a hepatic abscess.

We have throughout spoken of the large or solitary abscess, that is because in the disease we are describing the hepatic abscess is single in three-quarters of the cases, but, as already mentioned, in a few there are two large abscesses, and in a still fewer number three or more large abscesses. Sometimes we see a single abscess and many smaller ones; these are pyæmic. Perhaps the source of infection is the large abscess, or it may be intestinal ulceration.

About 80 per cent. of all large abscesses in the liver are in the right lobe, mostly at its upper and posterior part. The size of the abscess depends upon the time during which it has been forming; if the patient lives long enough, it may contain several pints—even seven—of pus. It is usually round, but may be of an irregular shape if two adjacent large abscesses have coalesced. Observers differ in their description of the liver substance in cases of large hepatic abscesses, but it is usually healthy.

The inner surface of the large abscess consists of shaggy granulation tissue undergoing superficial necrosis. When the abscess gets near the surface of the liver it sets up local peritonitis, and so the part of the liver in which the abscess is becomes adherent to surrounding parts; thus, when the

abscess is in the lower part of the anterior surface, the liver becomes adherent to the abdominal wall, or if the abscess is on the upper posterior part of the liver, adhesions form between it and the diaphragm; adhesions may form between the liver and some part of the alimentary canal, usually the colon. The abscess may even burst into the alimentary canal. The inflammatory process may spread right through the diaphragm, and then the adhesions form between the lower surface of the right lung and the diaphragm; so, as adhesions have already formed between the liver and diaphragm, the abscess may burst into the lung without infecting the peritoneum. Adhesions around a hepatic abscess may, if they last long enough, become very thick and hard. I have known them cut like gristle. Adhesions may, even after the abscess has been drained, give rise to much pain. The colour of the pus in a hepatic abscess depends upon the admixture of broken-down hepatic tissue; if there is none of this it is usually thick in consistence and pale yellow or greenish-yellow in colour, but when tinted by broken-down hepatic tissue, as it commonly is, the colour is that of anchovy paste. This is very characteristic of hepatic abscess. Sometimes blood is mixed with the pus.

If the abscess is associated with amœbic dysentery, amœbæ may be found in the pus or, more easily, on the inner surface of the abscess. Amœbæ may be

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found even when no clinical symptoms of dysentery have been observed and no dysenteric ulceration is found post-mortem. Such cases suggest that amœbæ may find their way to the liver, even when there is no intestinal ulceration. Bacteria may be present in large abscesses in the liver, streptococci, staphylococci, bacillus coli, and bacillus pyocyaneus are most often found, but if the abscess has existed some time the pus is often sterile. Amœbæ are found not only in the pus, but also in the wall of the abscess, the adjacent liver, and the minute terminations of the portal vein. The smaller, and therefore the more recent, the abscess the more numerous the amœbæ. Of course, the patient may chance to die while the abscess is still small; this variety of abscess is only called the large variety, because the patient usually lives long enough for it to become large or very large; he does not live long enough for this if the abscess is of the pyæmic variety. It is probable that the soluble products of the amœbæ cause local necrosis of the liver cells, and this is how the formation of the abscess begins.

Symptoms.—These are general and local. Fortunately for diagnosis the general symptoms are nearly always slow in onset, so that we are not likely to operate upon a case of portal pyæmia under the belief that the patient is suffering from a large abscess, for the illness in portal pyæmia generally quickly ends fatally. The most important general

symptom is pyrexia, and in very many cases this is the initial symptom. At first the rise of temperature is slight and irregular, but gradually it becomes characteristically hectic, with a wide excursion between the comparatively low temperature in the morning and the high temperature, it may be 103 or 104, in the evening. There are few diseases, except malaria, with wider or more regular daily excursions. This fact, together with the history of residence abroad, often led, before we knew how to find the malarial parasite in the blood, to an erroneous diagnosis of malaria. I remember a patient in whom there were no local signs of disease of the liver—he himself was sure he had malaria—and it was not until a post-mortem examination that the cause of his illness was found to be an abscess in the liver. The only consolation was that he was so weak and emaciated when he came into the hospital, and the abscess was so far in the liver, that it was very doubtful whether any operation could have saved him. It is stated that, even in the tropics, considerable intermissions during which no fever is present may occur, and that is certainly true of cases as we see them here in England. I remember the case of a middle-aged man, who about once every three months would suddenly feel ill, and then his temperature was always found to be raised; it continued to be of a hectic character for a few days, when it returned to normal, and he became

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well again. He saw many physicians, among others those with special experience in tropical diseases, for he had years before been in the East Indies, but none did him any good or suspected hepatic abscess ; but at last one, by carefully percussing the hepatic dulness, detected a small increase upwards in the axillary line, a needle was inserted, pus was withdrawn, and at a subsequent operation pus was evacuated from the liver. I have never seen a hepatic abscess unaccompanied by a rise of temperature ; but it is said that very rarely there may be no pyrexia, and in a few cases the pyrexia is not hectic, but constantly raised or irregular.

Rigors may be very striking and severe, thus increasing the resemblance to malaria ; in cases of doubtful diagnosis they are very suggestive of hepatic abscess. There are all degrees of severity of the rigor ; it may be represented by a slight shivering or a mere feeling of coldness, or the rigor may be so severe that the bed shakes. The patient often complains of profuse sweats which come on when he drops off to sleep. The sweating varies much ; it may be absent, or the patient's pyjamas may be soaked by it. The pulse is usually rapid in proportion to the temperature, and if the patient is very ill it is small and feeble, but if deep jaundice is present it may be slowed.

In bad cases the patient is excessively ill, weak, anæmic, and wasted to a mere skeleton. In this

country we see such cases on their arrival from India, and their illness has probably been made much worse by the discomforts of the voyage; indeed, I have known such a patient die shortly after arrival. Some patients, especially if there are long intervals between the periods of pyrexia, do not suffer much from impairment of their general health. Such probably are suffering from a comparatively small abscess with very thick walls. Between these two extremes there are all grades.

Often the blood shows a considerable increase of polymorphonuclear leucocytes. When this sign is present it is of the greatest possible aid to diagnosis, for it serves to distinguish a hepatic abscess from malaria. It is said that the leucocytosis remains constant, not fluctuating with the temperature. Unfortunately it is often absent, especially if the pus is sterile or the abscess has very thick walls. During the fever the patient is thirsty, has a poor appetite and a dry tongue, the urine is scanty and high-coloured, and may contain a trace of albumen. He may be constipated, but, owing to the frequent association of the disease with dysentery, diarrhoea is not uncommon. Vomiting, when present, is perhaps more a local than a general sign, for it is most frequently seen when the abscess is in such a position that it is in contact with the stomach. Swelling of and pain in the joints, presumably due to the absorption of toxins from the abscess, may occasion-

ally occur. If the case is fatal the patient usually sinks from exhaustion ; rupture into the peritoneal cavity is very rare. Finally, it should be added that occasionally a hepatic abscess is found after death in those in whom it is unsuspected during life, but as our means of accurate diagnosis increase such cases must become less common.

Among the local signs by far the most important are those obtained by physical examination of the liver. Hepatic abscesses are commonest at the upper part of the right lobe ; as they come near the surface of the liver adhesions form between them and the diaphragm, which becomes softened by spread of the inflammation, consequently the abscess pushes a part of the diaphragm up, and hence we get a dome-shaped area of dulness, varying in size from a shilling to several inches across, added to the top of the normal line of hepatic dulness. This line should in all cases be carefully mapped out with a blue pencil on the right chest, and then the additional local dulness upwards will be easily seen ; it is usually posterior to the mid-axillary line, often in the posterior axillary line, or even further back. The next most important part to examine carefully is the subcostal area in the right side ; sometimes when the patient draws a deep breath a rounded swelling may be felt on the surface of the liver, or the hepatic dulness may be increased downwards. An abscess has to be of considerable size to form

a visible swelling below the ribs, and still larger to push the ribs outwards and form a swelling of the side of the chest; it may be that without any actual swelling we may see, in a good light, that the depressions indicating the intercostal spaces on the right side are obliterated, or we may be able to feel this on passing the hand over the side of the chest. It must be by no means inferred that if there is no localised swelling the abscess is small; very large abscesses in the liver may exist and yet cause no local swelling. Local tenderness is very important. The whole of the hepatic region should be pressed by one finger, and not infrequently a localised tender spot will be found usually in an intercostal space; this is often a guide to the locality of the abscess. I have known no other localising sign to be present, yet a needle inserted at the tender spot withdrew pus. Inasmuch as the abscess is often under the ribs, rigidity of the rectus abdominis—often a great help to the diagnosis of abdominal disease—is frequently absent, but if the abscess presents towards the abdomen it may be present.

There is usually some pain in the hepatic region at some period of the illness, but it is very variable; it may be only a dull ache, or the patient may complain of a feeling of weight, distension, or dragging. When the formation of pus is rapid, the pain may be throbbing. Again, it may be very severe and stabbing. It is supposed that then the capsule

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of the liver has become implicated in the inflammation. Often coughing or drawing a deep breath makes the pain much worse. In such a case the breathing may be rapid and shallow, and the air may not enter the right lung as well as the left. Sometimes, if the patient is shaken in the same way as when Hippocratic succussion is obtained, pain may be caused. As with many other hepatic conditions, there may be pain in the right shoulder, it is said, in one-sixth of the cases; and if the left lobe of the liver contains an abscess, the pain may be in the left shoulder. But although all these varieties of pain may be very evident while the abscess is forming, yet some of the latent hepatic abscesses, which we see in this country among those who have been in the tropics formerly, are quite painless at the time at which we see them, and unless this is borne in mind they may be overlooked. Such abscesses often have a very thick wall. Authors, describing the acute cases seen in the tropics, lay stress on the fact that patients are seldom able to lie on either side without pain, and therefore lie on their backs in bed. But here again it is different with the chronic cases we see in this country among those who have been abroad, it may be many years before. Often such patients are able to walk about very well without any pain at all.

Edema and redness of the skin and fluctuation can of course only be present when the abscess

is near the skin. Well-marked jaundice is only present in the rare cases in which the abscess chances to press upon the hepatic duct; but a few patients have a slightly yellow tint, which has been assigned to slight catarrh of the bile ducts. The abdomen is sometimes full and distended; this is usually due to flatulence, but sometimes to hepatic enlargement, for the liver as a whole may be enlarged and project some distance below the ribs.

In any case of the slightest doubt, the patient should be X-rayed; for not only may the abscess itself be in such a position that it will throw a shadow, but the impaired movement of the diaphragm on the right side may show that there is disease in its immediate neighbourhood.

The duration of the case, if untreated, is usually about four months, but very acute cases have been known to be fatal in a week or two. The most difficult cases to diagnose are those mostly seen in this country—in which the patient has not been in the tropics for many years, it may be twenty. Whether we are in such cases to suppose that the abscess did not form till many years after he left the tropics, or whether, as perhaps is more likely, an abscess which formed in the tropics has for years been quiescent and then gives rise to symptoms, we do not know. But it must in actual practice be borne in mind that if a man has ever been in the tropics he may, although he has not resided there for many

years, show without apparent cause, symptoms of abscess of the liver, and such should always be most carefully looked for when he has pyrexia, the reason for which is not apparent. In such a case the abscess is, as far as my experience goes, usually comparatively small, and therefore the physical signs must be gone over with the greatest care, and it often has a thick wall. I have seen the wall so thick and tough that the surgeon could hardly cut it. This thickness of the wall may explain the slightness of the symptoms in these cases, for it may greatly impair the absorption of toxins from the abscess.

I once saw a man who had had, years before when in the East, an abscess which had been drained. While in Europe he was taken with a febrile illness, so slight that some of those who saw him would not believe his statement that he was sure he had another hepatic abscess; but such was his persistence that the liver was explored with a needle, pus was found, and the abscess drained. And I have seen two or three cases in which both the general and local symptoms, although they suggested to the doctor himself that an abscess was present, have been so very slight that they might well have been overlooked.

Termination.—If the abscess is not drained the patient usually dies of exhaustion, but the abscess may burst spontaneously. Putting together Cyr's

and Thierfelder's 329 cases, we find that the abscess burst into the lungs in 133, into the pleura in 57, into the pericardium in 5, into the peritoneal cavity in 62, into the stomach in 21, into the intestines in 41, externally in 2, and in the remainder into the kidney, inferior vena cava, or bile passages. In each of six cases perforation occurred in two positions.

Accurate conclusions cannot, however, be drawn from these figures, for it takes no account of the cases for which operation was done, nor of the cases which died and did not rupture; and, further, judging by the fact that a patient whose appendicular abscess bursts into the bowel usually gets well, we may infer that some of those whose hepatic abscess bursts into the gastro-intestinal tract recovered, and thus would not appear in the above figures. Even rupture into the pericardium is not necessarily immediately fatal, for I once saw a man in whom the history and state of affairs found at the post-mortem pointed to the fact that rupture of an abscess of the left lobe of his liver into the pericardium had occurred a month before he died. This was a case of great interest for several months before he died. An abscess of the liver was so strongly suspected that the right lobe was needled under an anæsthetic many times and no abscess was discovered. The post-mortem showed the right lobe to be healthy. The abscess was at the top of the left lobe. Only

about 10 per cent. of all hepatic abscesses are confined to the left lobe. Rupture into the lung can hardly take place without some spread of the inflammation to the lung, so that we may consider it under the next heading.

Complications.—By far the most important are pulmonary. An abscess of the liver, being commonest at the upper part of the right lobe, soon causes inflammation of the diaphragm as it comes to the surface. Thus an irritating cough, without any expectoration, is a common early sign of hepatic abscess; deep breathing causes pain; the movement of the chest on the right side is impaired. The deficient movement of the diaphragm is easily seen on the screen when the patient is examined with the X-rays. As the inflammatory process spreads, a basal pleurisy ensues and adds to the pain of deep breathing and coughing, and still further diminishes the movement of the diaphragm. If the pleurisy spreads far enough to the side it causes an audible rub; the breathing is shallow, rapid, and diminished on the right side. It must be carefully borne in mind that patients sometimes first seek the advice of a doctor for these pulmonary troubles, and only with difficulty is it made out that they are really secondary to a hepatic abscess. The next stage is that the base of the right lung becomes solid; this is partly due to compression and partly to pneumonia. The usual signs of solidification of the lung may be

discovered on physical examination; but often they cannot be made out because the solidification is so deep-seated, and even if physical signs are present it may be difficult to determine how far they are due to the actual abscess in the liver, which as it grows pushes the softened diaphragm upwards. The pleurisy at the base of the lung that commonly follows a hepatic abscess leads to dense adhesions. It is this fact that renders it possible for us often to reach a hepatic abscess with a needle or a knife without opening the pleural cavity. Soon the abscess bursts through the diaphragm; if by that time the adhesions between its upper surface and the lung are very slender or have not formed, the pus passes into the pleural cavity; but usually rupture takes place into the lung itself, because at the point of rupture the pleural cavity is obliterated by adhesions.

We then have an abscess in the lung which, as the lung tissue is loose, soon increases in size. Usually the aperture through the diaphragm is small, so that the abscess in the liver and that in the lung form an abscess the shape of an hour-glass. This should be remembered when we are trying to drain such an abscess by an external wound. Sooner or later the abscess in the lung ruptures into a bronchus and pus is coughed up. The expectoration is purulent and nearly always some shade of red; for, in the first place, the pus of a hepatic

abscess is often of an anchovy paste colour; and, secondly, the congested lung in the neighbourhood of the abscess readily bleeds. Even after the hepatic abscess has healed, that in the lung may remain, and the expectoration may contain amœbæ and continue to be red.

Rarely, if a hepatic abscess opens into a bile duct as well as the lung, the expectoration may be bile-stained. Occasionally more than one abscess forms in the lung, and the pulmonary abscess may be of such a shape that drainage of it may be very difficult.

It is well known that a cerebral abscess occasionally occurs as a complication of an empyæma or an abscess in the lung; hence it is not surprising that they have, in a few instances, been known to follow hepatic abscesses, and it would be of interest to know whether they ever occur as a complication of a hepatic abscess in cases in which there is no pulmonary abscess. The cerebral abscess does not contain amœbæ.

There are no other special complications. Rupture into the peritoneum causes fatal general peritonitis. Rupture into the stomach or duodenum causes vomiting of pus, and if the abscess is large some may be found in the motions. Pus is naturally more likely to be found in the motions in cases in which the rupture takes place into the colon. Rupture into the pleural cavity gives rise to an empyæma.

Prognosis.—This depends to a large extent upon the associated conditions. Thus patients who are already severely ill from dysentery or from over-indulgence in alcohol do badly. If portal pyæmia has resulted from the abscess, the outlook is hopeless. If, too, more than one abscess is present the outlook is serious. Patients may be successfully operated on for one abscess, and later on, it may be years after, another may form. I saw in this country a man who had lived in India many years; he went to Egypt, was there successfully operated upon for a hepatic abscess; he was successfully operated on, for a second, in this country; he went abroad again, but not to the tropics; when he returned to London, a long while after, a third abscess had formed; this burst both into his chest, giving him a pneumo-thorax, and into his bowel, but he ultimately got quite well and has remained so. There is no doubt that this man was exceptionally fortunate; but it is not cases like his, in which the abscesses succeed one another, that are so serious as those in which more than one abscess is present at the same time, for then the second abscess is often overlooked; for example, I saw a man who had been in the tropics and in whom the surgeon had successfully opened one hepatic abscess. From the temperature chart it appeared that another must be present, although there was no leucocytosis; the patient was most thoroughly explored under an

anæsthetic for the second abscess, but none was found. One day, however, there was a gush of pus from the bowel, and the temperature dropped to normal; no doubt the second abscess had ruptured into the colon. If, as is sometimes the case, the abscess is of a very irregular shape, so that efficient drainage is difficult, the gravity of the outlook is increased. But if a single abscess is detected soon after it has formed, and the patient has nothing else the matter with him, the outlook is good if it is properly drained by operation.

We see in this country a considerable number of patients who are invalided home because a hepatic abscess has burst into the lung. Like empyæmas which burst into the lung, they do better than might be expected. The prognosis depends chiefly upon whether the hepatic abscess or the empyæma becomes the source of a general pyæmia, and it is by this that the prognosis must be judged. If a hepatic abscess bursts into the colon the patient usually recovers, as do those who suffer from an appendicular abscess which bursts into the bowel. Often those hepatic abscesses which lead to an empyæma or pulmonary abscesses are difficult to drain properly, for the communication between the hepatic abscess and the chest may be small and difficult to find. In such a case the prognosis should be guarded. Those that burst into the peritoneum, pericardium, or the vena cava, or vena

portæ are fatal. In the rare instances in which one bursts externally the patient will probably get well.

Diagnosis.—No useful purpose would be served if we were to discuss all the possibilities of mistake in connection with a hepatic abscess. Hepatic abscess and malaria have been confused; the examination of the blood will prevent this.

Suppuration about the liver—subphrenic abscess—may readily be mistaken for hepatic abscess, but the history in the two cases is quite different (see p. 114). Physical signs occur much earlier, as a rule, in the case of a subphrenic abscess; the physical signs may be those of air and pus in the case of a subphrenic abscess, and a coin sound may be present; the liver does not usually descend when a subphrenic abscess is present, for the extensive adhesions between the hepatic and parietal peritoneum prevent this, but a hepatic abscess may so enlarge the whole liver that its lower border descends considerably. The pus from a hepatic abscess is usually like anchovy paste in colour; it may contain amœbæ with or without other micro-organisms, or it may be sterile; but that from a subphrenic abscess is yellow and contains micro-organisms, but not amœbæ. Then, too, a subphrenic abscess is subphrenic, but a hepatic abscess need not be. Subphrenic abscess is not always confined to men, but tropical abscess almost always is. Leucocytosis is a more constant feature of subphrenic than hepatic abscess.

Portal pyæmia may cause a difficulty, but this is usually a much more rapid disease than tropical abscess; the genenal symptoms are usually more severe; those of any localised enlargement of the liver are rare, and jaundice is more common. The X-rays may be of help.

Gall-stones accompanied by rigors may be confused with a hepatic abscess; but if they are in the common duct, jaundice will be present, which is not often the case when a patient has a hepatic abscess; and again, there will be no physical or X-ray signs of a hepatic abscess and no leucocytosis, but there probably will be a history of previous attacks of jaundice, but usually none of residence in the tropics, and gall-stones are common in women. A suppurating gall bladder and a suppurating hydatid may be difficult to tell from a tropical abscess, but usually in the former case, and often in the latter, a careful consideration of the physical signs and history will enable us to come to a correct diagnosis.

I have, more than once, known malignant disease of the liver thought to be an abscess in the liver because of some leucocytosis and hectic temperature; but a mistake may usually be avoided by a proper appreciation of the feel of the enlarged liver, and by a careful search for other evidence of malignant disease. The confusion is perhaps most likely to occur when gall-stones have induced malignant

disease of the gall bladder or bile ducts, for the gall-stones may give rise to rigors, and rapidly growing malignant disease anywhere in the body, and perhaps especially in the liver, may cause pyrexia and leucocytosis.

Tropical congestion of the liver (see p. 68) may resemble hepatic abscess, but after a few days' observation the distinction becomes clear.

Syphilitic pyrexia has, in India, been thought to be due to hepatic suppuration, but the mistake could hardly occur in this country. The pyrexia of tuberculosis, that of malignant endocarditis and that of Hodgkin's disease have each been thought to indicate hepatic suppuration. Pancreatic abscesses and abscesses in the abdominal wall have been confused with hepatic abscess.

It is clear that, as a hepatic abscess often gives rise to pleurisy, consolidation of the base of the right lung, and empyæma, it may sometimes be difficult to tell whether these conditions do or do not owe their presence to a hepatic abscess. When they do it is sometimes not easy to find the abscess, for if it is small and the amount of pleural thickening set up by it is extensive and of long standing, as is often the case with patients seen in this country, we find the lower part of the chest dull over a large area; but although from the history, temperature and leucocytosis, we may feel pretty confident that an abscess is present in the

liver, yet we hardly know where to explore. I have known the chest to be explored in over a dozen places, under an anæsthetic, before the abscess was found. The X-rays are no use in such a case, for the extensive pleural adhesions obscure everything; the best thing is to begin the exploration where the dulness is most striking, where the entry of air is least, and especially where there is a local spot of tenderness, if one is present. By patience and careful physical examination the abscess can usually be found, if it is present. The ideal proceeding is to give an anæsthetic and be prepared to operate, while the patient is under the anæsthetic, should pus be found. If this is done, an ordinary aspiration needle may be used. It should be passed in for three inches and then slowly withdrawn, so that if it has passed through the abscess pus will flow into it as it is withdrawn. There is a slight risk that if operation is not done directly pus is found, the pus may pass through the opening made by the needle into the pleural cavity and cause an empyæma; but when, as in the case we are considering, the pleural adhesions are very extensive this risk is hardly appreciable, and in such a case, as a matter of practice, the chest is often needled without an anæsthetic. Still, whenever possible, this should be given and the operation proceeded with at once, if pus is discovered. As the abscess has grown up from the liver the needle,

as a rule, should be passed inwards and a little downwards. It should very rarely be passed more than three inches, because if it go too far a large vessel may be wounded. It need hardly be added that a needle should not be passed through the abdominal wall to search for an abscess, for the pus will very likely leak into the peritoneal cavity and cause general peritonitis.

Treatment.—In a case of uncomplicated hepatic abscess the pus should be let out and the details of the operation will be found in books on operative surgery. If the abscess has ruptured into the bowel, surgical interference is rarely necessary. The most difficult cases that occur in this country are those in which a patient has been invalided home because an abscess is discharging through the lung. As in an empyæma discharging through the lung, so in these cases the chief guide as to whether an operation should be done is whether the patient is in a thoroughly septic state from the absorption from the abscess. Often he is not; the abscess having very thick walls, absorption of toxins and bacteria is slow. The mere change of air to this country often does much good, and the patient should certainly be watched for at least a fortnight before operation to see whether he improves without, for when these patients first land here we see them at their worst, owing to the discomforts and fatigue of the voyage. My experience is that by fresh air,

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especially at the seaside, good feeding, and nursing the discharge of pus by the lungs nearly always slowly gets less, and the patient gains in weight and strength. Not long ago I saw a man who was landed at Tilbury so ill that the doctor who met him doubted whether he would survive the journey to his house in the suburbs. His temperature was raised, he was wasted to less than half his normal weight, and he coughed up a pint and a half of anchovy paste pus in twenty-four hours. There was a discussion as to whether he should be operated upon, but it was decided that he should not, for some felt he would probably die under the operation. At the end of two months he had gained three stone in weight, his temperature had been normal for a month, and he only coughed up a few ounces of pus in twenty-four hours. He then went to the seaside, and five months after landing he looked so well that he was unrecognisable; he weighed nearly twelve stone, could walk long distances, and once in three weeks or so he would get a slight rise of temperature, and this would fall when he had coughed up a little pus. Now this has stopped, and he is perfectly well. I know a man who for many years past has been perfectly well except that about twice a year he has a temperature for a day or two; he then coughs up a pellet of anchovy paste-like pus and is better. I have under observation two patients in whom a

vaccine has been prepared from the micro-organism present in the pus discharged, and injection of this vaccine has benefited both, and in one expectoration has entirely ceased.

So long as the patient, whose hepatic abscess is discharging through the lung, is improving, it is wise not to search for the abscess or to operate, for in the first place, such an abscess is very difficult to find, because usually it is surrounded by very extensive thickening of the pleura; in the second place, it is often deeply situated and perhaps of irregular shape, so that it is difficult to reach and drain, and the operation may be very severe; and thirdly, as by coughing the abscess is repeatedly emptied, an exploring needle may easily fail to find pus. But if from the persistence of hectic temperature, profuse sweats, loss of weight, and other signs of general infection, it appears that the patient is losing ground, then an attempt must be made to drain the abscess which is discharging into the lung. It is a good thing, if it is determined to explore, to give the patient in the evening enough morphia subcutaneously to prevent coughing during the night; the pus then collects and may be reached by an exploring needle.

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SUPPURATION AROUND THE LIVER

THE consideration of this hardly falls within the scope of diseases of the liver, but as confusion may arise between abscesses outside and abscesses inside the liver, I will give briefly the chief symptoms of the common varieties of those in the neighbourhood of it. As the treatment is entirely surgical, nothing will be said about that.

It is not excessively uncommon to find abscesses outside the liver but in contact with it. By far the most important clinically are those between the diaphragm and the liver. They are usually called subphrenic abscesses.

Subphrenic Abscess.—In this country about a quarter of all subphrenic abscesses are due to a gastric ulcer which has caused adhesions to form on the outer surface of the stomach ; perforation takes place into these adhesions and an abscess forms, which tracks up under the liver. One-fifth are due to a hepatic abscess—sometimes tropical, sometimes a suppurating hydatid—which comes to the surface of the liver, and so completely thins out the covering of hepatic tissue that a subphrenic abscess forms, the

diffusion of the pus generally being prevented by adhesions. Between a sixth and a fifth are due to appendicitis. A duodenal ulcer is the cause in almost seven per cent. of the cases. The other causes, though numerous, are each very rare. They will be found given in an article by Mr. H. L. Barnard. Although cases in which a subphrenic abscess has followed suppuration in the chest, *e.g.* empyæma or bronchiectasis, have been recorded, they are quite exceptional. Pus hardly ever travels downwards through the diaphragm; but, on the other hand, it often travels upwards, and an empyæma may follow hepatic abscess, subphrenic abscess, or appendicitis, or, indeed, any intra-abdominal suppuration. The reason for the frequency of abscess between the liver and diaphragm is chiefly that ulcers of the stomach are commonest at its pyloric end, abscesses of the liver are commonest at its upper part, the appendix and duodenum are on the right side of the body, but also because, as the patient lies in bed, the space above the kidney is on a lower level than the kidney, the eminence of which forms the lower boundary of a depression in which pus easily collects.

Often the abscess is limited by the folds of the peritoneum which form the ligaments attaching the liver to the abdominal wall; thus, as might be expected, considering that the usual place for a gastric ulcer is near the lesser curvature towards

the pyloric end of the stomach, an abscess connected with a gastric ulcer is usually a left anterior intra-peritoneal subphrenic abscess, or, as it is sometimes called, an anterior peri-gastric or a perisplenic abscess. All these names refer to its position, for the left lobe of the liver is below, the spleen to the left, the suspensory or falciform ligament of the liver to the right, and the diaphragm above. The abscess is limited behind by the left lateral ligament of the liver, and in a downward direction by adhesions between the stomach and anterior wall of the abdomen. When an abscess is in this space it rarely passes into any other.

A subphrenic abscess due to appendicitis is usually on the right side of the falciform ligament; if, as is rather more often the case than not, it is in front of the right lateral ligament, it is a right anterior intra-peritoneal subphrenic abscess, bounded to the left by the falciform ligament, below by the right lobe of the liver, above by the diaphragm to the left of the falciform ligament, behind by the right lateral ligament, and in front by adhesions between the anterior edge of the liver and the abdominal wall.

If the abscess is behind the right lateral ligament, it becomes an intra-peritoneal right posterior abscess; the fossa in which it occurs is known as the subhepatic pouch or right renal fossa. Its right-hand end is the most capacious, and is formed by the abdominal wall, and the fossa

reaches just below the last rib. With this right-hand end as a base, the fossa is triangular, with apex to the left. The liver and gall bladder form the anterior boundary, the posterior is formed by the upper part of the right kidney, the lower part of the diaphragm and its crus, by the duodenum and bile duct; then comes the eminence of the spine dividing the fossa into right and left parts; the left is very narrow, and is bounded by the left lobe of the liver in front, and the small omentum and the anterior wall of the stomach behind. Above right renal fossa is bounded to the right by the right lateral ligament of the liver, and to the left by the transverse fissure. Below the boundary is formed on the left by adhesions between the anterior surface of the stomach and the margin of the liver, and on the right by adhesions of the great omentum and transverse colon to the margin of the liver and anterior abdominal wall. This fossa communicates at its base with the right anterior subphrenic space, and when it is infected the right anterior subphrenic space is nearly always infected also. It appears that when an appendicitis infects the right posterior space suppuration spreads to the right anterior space much more often than it spreads from the anterior to the posterior when the anterior is primarily affected. The right renal fossa is connected by the foramen of Winslow with the small sac of the peritoneum.

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An hepatic abscess, when it forms a subphrenic abscess, usually does so in the right-hand part of the space between the layers of the coronary ligament—that is to say, it is a right extra-peritoneal abscess; if not there, the abscess is almost sure to be in the right anterior intra-peritoneal space.

The formation of an abscess due to a gastric ulcer in the left anterior intra-peritoneal space, of an abscess due to appendicitis in either the anterior or posterior right intra-peritoneal space, or of an abscess derived from a hepatic abscess in the extra-peritoneal space or the right anterior intra-peritoneal space accounts for by far the greater number of subphrenic abscesses; indeed, the only other space is the left intra-peritoneal posterior, and that is rarely affected. But it is very important to remember that subphrenic abscesses often spread from one space to another, or, indeed, track far beyond their original seat; it may be down into the pelvis or up into the chest, so that each case requires most thorough physical examination; and if after one abscess is opened the temperature and leucocytosis do not fall, the most careful search must be made for another abscess. The reader will find much information about the association of the various abscesses in Mr. Barnard's article. To show how the process may spread, he gives the case of a man in whom a pelvic abscess, originally around the rectum, formed a track in the fossa to the left

of the lumbar vertebræ, then a left subphrenic abscess formed; this led to a left empyæma, which discharged into a bronchus, so that pus connected with a pelvic abscess was expectorated.

Symptoms.—As might be surmised from the frequency of gastric ulcer and appendicitis as causes of subphrenic abscess, it is commonest between the ages of twenty and thirty, and next commonest between thirty and forty. It is equally common in the two sexes. This may at first sight cause surprise, for it is often stated that gastric ulcer is much commoner in women than men, but I have elsewhere given reasons for doubting this, and have shown that many women thought to be suffering from gastric ulcer are really afflicted with gastrostaxis. There are, of course, often, but not necessarily, symptoms of a previous gastric or duodenal ulcer in cases in which the abscess follows either of these ulcers; if due to a hepatic abscess, we often have a history of residence in the tropics, and frequently the sufferer has had dysentery. When the abscess is due to appendicitis, there may or may not be a history of previous attacks; usually not, for if an abscess is going to form as a result of appendicitis, it generally does so in the first attack. There may be local evidence in the right iliac fossa of appendicitis; but I have known this absent when the appendix has been very long and directed upwards, for the appendicitis has taken place at its extremity.

The onset of symptoms is sudden in half the cases, which is not surprising, as subphrenic abscess is mostly due to perforation of a gastric ulcer or the appendix. Pain is very common, and is always felt in the situation in which the abscess forms. Vomiting occurs in most cases. The temperature is raised, and is higher in the evening than the morning. The degree to which it is raised varies between 100 and 105. The pulse is rapid. Leucocytosis is almost always present, and is a very valuable sign. The tongue is dry, the face pale, profuse sweats are common. Rigors may be severe, but are often absent; cases in which they are present usually do badly. At first the bowels are generally constipated, but later on, as the patient becomes septic, diarrhœa supervenes. This is a bad sign. The bursting of an abscess into the bowels causes diarrhœa. I have known diarrhœa due to this to be so continuous as to exhaust the patient considerably.

In a considerable majority of the cases there is, after a few days, a visible tumour, and generally it is caused by the abscess itself; but when the abscess is primarily in the liver the swelling, which is due to general enlargement of the liver, may be lower than the abscess. The swelling may be of the abdominal wall or the lower thoracic wall. A tape measure usually shows the chest to be larger on the affected side. As in the case of liver abscesses

local tenderness, which may be discovered by the finger, is often to be detected, and when present is a very valuable sign, not only of pus but also of its locality. If the abscess is coming towards the abdominal walls there is usually muscular rigidity over it. If a swelling is present it is dull and occasionally fluctuation may be obtained. A subphrenic abscess can often be seen with the X-rays, for it casts a very dark shadow. Occasional signs are local cedema, local enlargement of veins, and a peritoneal rub. It is particularly to be remembered that the liver is not pushed down by a subphrenic abscess, for a localised abscess could not be formed unless adhesions took place between the liver and surrounding structures or the abdominal wall, and these adhesions prevent the displacement downwards of the liver.

When perforation has taken place from the stomach or duodenum, in a few cases, but not nearly so many as is commonly supposed, the abscess contains air as well as pus. The air, as the patient lies in bed, rises uppermost, and if the abscess is in front of the lateral ligaments, as it usually is, there is a resonant area, over which a *bruit d'airain* can be heard by placing one coin on the skin and hitting it with another coin while listening with a stethoscope. As the patient moves the air moves, and hence these signs shift. Very rarely splashing may be heard on listening to the seat of abscess while the patient is

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shaken, as in obtaining Hippocratic succussion in a pyopneumo-thorax.

As might be expected, signs connected with the chest are often seen. Thus cough is often present, signs of compression or consolidation of the lung may be discovered ; or, again, there may be a collection of fluid, sometimes clear, sometimes purulent, in the chest. Thus it is not surprising that dulness over the lower part of one lung or the other is common, and bronchial breathing may be heard, with increased bronchophony if the lung is solid, or a diminished air entry with diminution of voice sounds and vocal fremitus if fluid is present. Sometimes a pleuritic rub is heard ; rales, rhonchi and crepitations may be discovered. Pericarditis is very rare, but the impulse of the heart may be displaced upwards by the subphrenic abscess, or laterally by the pleuritic effusion. An empyæma may be met with in a few cases. If the subphrenic abscess is to the right of the suspensory ligament or in the right-hand part of the space between the layers of the coronary ligament, the thoracic signs will be in the right chest. If it is to the left of the suspensory ligament, or in the left-hand part of the space between the coronary ligament, the thoracic signs are often in the left chest.

If a subphrenic abscess is suspected we should not be in too great a hurry to explore with a needle or to operate. I have always taught this, for I have seen

delicate adhesions ruptured in the course of early operation, with the result that general peritonitis has supervened and the patient has died. Further, when the abscess is small it is far from the surface, and therefore difficult to reach and difficult to drain. The abscess should not, as a rule, be sought for till between the tenth and fourteenth days after the onset of symptoms, and this is true not only of sub-phrenic but also of many other intra-abdominal abscesses. When it is determined to seek for the abscess an anæsthetic should be given. If the position of the abscess is such that it has to be opened through the abdominal wall an exploring needle should not be used, for the peritoneum may be infected with pus if it is; but in many cases exploration has, from the position of the abscess, to take place through the intercostal spaces, then generally a needle may be used without the fear of infecting the peritoneal cavity. Usually pleuritic adhesions have obliterated the lower part of the pleural cavity, or the abscess has so pushed up the diaphragm that it is in contact with a large area of the lateral chest wall, and so the pleura is not infected by exploration. When the patient is under the anæsthetic he passes into the hands of the surgeon. A full and admirable account of the best situations for puncture, and the best operations to adopt according to the various positions of the abscess, has been recently

given by Mr. Barnard, from whose paper many of the facts in this short article have been derived. It is important never to use an exploring needle unless the patient is under an anæsthetic and all arrangements have been made for continuing the operation if pus is found, for very likely many painful needlings will be necessary before pus is found; and if the needle, after pus is found, is taken out, pus may leak along its track and burst into or infect the peritoneum or the pleura.

Cure may take place without operation, but such an event is so rare that in almost all cases the attempt should be made to drain the abscess surgically. Among 23 cases in which the abscess ruptured spontaneously, it burst into the stomach in 8 cases, into the bronchus in 4, into the left pleura in 3, into the right pleura in 2, into the colon in 2, through the skin in 2, into the general peritoneum in 1, and into the intestine in 1.

The difficulties of diagnosis are often great. It is commonly taught that a subphrenic abscess pushes up the part of the diaphragm in contact with the abscess, and that therefore we have upon the top of the normal hepatic dulness a local dome-shaped dulness of greater or less extent according to the size of the hepatic abscess. In some cases this can be mapped out by careful percussion, and is then of very great value in enabling us to come to a decision; but unfortunately in many cases

there is either fluid in the pleura or consolidation of the lung, either of which completely obliterates the dulness due to the subphrenic abscess. Again, it is often taught that a subphrenic abscess depresses the liver, but it is quite exceptional for it to do so, for a subphrenic abscess would not be present unless there were peritoneal adhesions in connection with the liver, and these prevent its downward displacement.

It is clear that confusion may arise between an empyæma and a subphrenic abscess, but an important distinction is that, because an empyæma usually makes room for itself by compressing the lung, it rarely causes a local bulging of the chest, or an increased measurement round the affected side as compared with the non-affected, but a subphrenic abscess frequently does both. Sometimes a screen examination with the X-rays helps us to tell whether the fluid is above or below the diaphragm, but the inflammation of it, whether caused by an empyæma or a subphrenic abscess, in either case greatly restricts its movement. If, as Acland says, on exploring the lower part of the chest a layer of clear fluid, with a layer of pus lower down is found, the case is probably a subphrenic abscess with pleurisy, rather than an empyæma. We must never forget that often in pleurisy and pneumonia pain is referred to the abdomen. Many a person has been thought to have an acute inflammation within the abdomen,

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when in reality the whole of the trouble has been intra-thoracic. When the abscess contains air, the diagnosis that it is subphrenic is usually easy, for a pneumo-thorax could hardly give a coin sound below the ribs.

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CIRRHOSIS OF THE LIVER

MANY descriptions of cirrhosis of the liver that have been written are extremely confusing. This is because the phrase is used in two senses. The histologist calls the liver cirrhotic whenever he can find a little, ever so little, increase of fibrous tissue in it. The clinical physician, however, restricts the term to the liver, which we all know so well on the post-mortem table—the hard liver with a great increase of uniformly distributed fibrous tissue, which is easily recognised by the naked eye. Cirrhosis which is only histological is of very little clinical interest. All that need be said about it will be given at the end of this chapter, and we are now concerned only with the clinical variety of cirrhosis.

Description of the Liver.—It is usual to describe two varieties of clinical cirrhosis, namely, that in which the liver is large, the so-called hypertrophic cirrhosis, and that in which the liver is small, the atrophic cirrhosis; both are hard from the increase of fibrous tissue. This nomenclature has been sanctioned by usage; but it may lead to confusion,

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for there is an extremely rare condition, known as hypertrophic biliary cirrhosis, which has nothing to do with that now being described, and of which an account will be found on p. 171. And sometimes a liver in which there has been a great replacement of hepatic tissue by fibrous tissue, and therefore it is in a sense atrophied, really weighs as much as, or more than, a healthy liver, although it may be smaller than normal, because fibrous tissue has a higher specific gravity than healthy hepatic tissue.

The hypertrophic or large cirrhotic liver may weigh as much as 150 or 200 oz., but this is exceptional; the weight rarely exceeds 100 oz. The increase of weight is due chiefly to increase of fibrous tissue, sometimes to compensatory hyperplasia of liver cells, many of which soon undergo fatty degeneration, and sometimes to congestion of the vessels. The liver is not altered in shape, and in the early stages the surface is smooth; but as the fibrous tissue contracts and the fat is absorbed from the cells which have undergone fatty degeneration, the surface becomes finely uneven. The colour is nearly always paler and yellower than that of a healthy liver. This is due principally to the fatty cells, but if the patient is jaundiced it is partly owing to staining with bile. The increase of fibrous tissue makes the liver tougher than in health.

The atrophic liver is small; it may weigh only 30 oz. The peritoneal surface is irregularly opaque.

This is sometimes due to a patchy perihepatitis, but more often to subcapsular fibrosis and atrophy of the liver cells. General perihepatitis is very unusual (see p. 203). The liver is deformed from the contraction of the fibrous tissue which cuts the organ up into small islets; and as these remain prominent, in contrast to the contracting fibrous tissue, they stand out, forming what are commonly called hobnails—each usually being about the size of a large hobnail, but all sizes smaller than this may be seen. Sometimes the fibrous tissue is more evident in the left lobe than the right, and then the left lobe is more atrophied than the right. The hobnails have been described as multiple adenomata (see p. 239). The liver is exceedingly tough, of a darker colour than the hypertrophic variety, for the fat in the fatty degenerate cells has been absorbed, and the staining with bile has lasted some time. On section the liver is seen to be divided up into a number of areas, each the size of a hobnail, or smaller, by narrow bands of grey fibrous tissue; each area contains between five and ten lobules, and hence this variety of cirrhosis is often called multilobular cirrhosis, and because the fibrosis spreads from the smaller portal canals it is also called portal cirrhosis. As the new fibrous tissue continues to grow, fresh processes enclose smaller areas of hepatic cells, and therefore, in some parts of the liver, the cirrhosis becomes monolobular; at a still later stage

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the lobules themselves are invaded, consequently in some sections the cirrhosis is seen to be intralobular.

Under the microscope, in addition to the fibrous tissue, we see lying among it columns of small cells, often cubical, staining deeply, and evidently in a stage of active growth. These are arranged in such a way as to suggest that they are newly formed branching bile ducts, and many writers have described them as such; but they are probably to be regarded as rows of recently developed liver cells which have been formed in an attempt to compensate for the destruction of other liver cells. In the earliest stages of cirrhosis there is no visible alteration in the hepatic cells, soon they undergo a compensating hypertrophy, and finally fatty degeneration. The bile ducts show nothing abnormal; the minute branches of the portal vein are sometimes compressed and very rarely thrombosed. Thrombosis of the portal vein itself occurs in about 3 per cent. of the fatal cases of cirrhosis.

There is considerable evidence that the enlarged liver gradually becomes smaller as the disease progresses. Certainly this is what we should expect, for abnormal fibrous tissue usually contracts as time goes on, the atrophy of the liver cells continues, and the fat in those which had undergone fatty degeneration becomes absorbed. Indeed, Dr. Frederick Taylor and others have recorded cases in which cirrhotic livers became smaller under obser-

vation. J. A. P. Price analysed 142 fatal cases of cirrhosis, and found that in 80 the weight of the liver was over 60 oz., in 33 it weighed between 50 and 60 oz., and in 29 it was under 50 oz. This alone shows the futility of trying to draw a sharp distinction between the hypertrophic and atrophic varieties, for in such a classification it would be difficult to find a place for those weighing between 50 and 60 oz. As in the above series more livers were over the normal weight than under, the figures strongly suggest that the cirrhotic liver is at first larger than normal, and then, among those patients who survive, shrinks so as to become of normal weight, and then, continuing to shrink, becomes of less weight than normal in those who still survive. The view here taken is supported too by the fact that those who have atrophic livers are, as a rule, older at death than those who have hypertrophic. Formerly the attempt was made to show that the symptoms of those who had hypertrophic cirrhosis were different from those in whom the liver was small; but it has been shown that such differences are few, and are to be explained by the fact that certain symptoms come on late in the disease, and naturally, therefore, would be met with mostly in those in whom the cirrhosis was atrophic.

Description of the Morbid Appearances of other Organs.—Before leaving the morbid anatomy of cirrhosis of the liver, it will be well to describe the

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other conditions found in the post-mortem room and directly associated with the cirrhosis.

The capsule of the spleen is often thickened ; usually the thickening occurs in local patches only, but if general chronic peritonitis is present, the whole of the capsule is thickened, and this may even be so when no general peritonitis is to be seen. The spleen is usually enlarged, and this is due to the dilatation of its vessels, and if this has persisted for some time the fibrous tissue increases and the spleen becomes hard. But often it is soft ; this is probably because of terminal bacterial infection, and then sometimes there is hæmorrhage into the splenic pulp. It is commonly stated that the enlargement of the spleen is due to backward venous pressure, arising from constriction of the minute portal veins in the liver by contracting fibrous tissue ; but this is not a sufficient explanation, for the spleen attains a much greater size in cirrhosis than in heart disease with backward pressure, although in a severe case of nutmeg liver the increased venous pressure must be considerable. The spleen may in cirrhosis be enormous. Dr. Frederick Taylor mentions one which weighed $87\frac{1}{2}$ oz., and among 64 cases in which the spleen weighed over 20 oz. 17 were instances of cirrhosis of the liver. Backward venous pressure may contribute to the splenic enlargement, but it is probably also due to some toxin acting directly on the spleen, for we shall see, when we have described the

symptoms of cirrhosis, that probably many of them are toxic. French writers often state that the spleen is largest in the cases in which the patient dies with an enlarged liver, but this is not so; there is no relationship between the size of the liver and that of the spleen.

The difficulty of the flow of the blood through the finer branches of the portal vein in the liver, which results from their constriction by the contracting fibrous tissue, leads to dilatation of the peripheral branches of the portal vein, especially where they form anastomes with the general venous system. Thus the small branches of the portal vein, which form a communication, at the lower end of the œsophagus and cardiac end of the stomach, between the œsophageal veins which open into the azygos veins, and the gastric veins, may be varicose and dilated, and so may the veins forming a communication between the superior hæmorrhoidal vein, which returns its blood by the portal vein, and the inferior and middle hæmorrhoidal veins, which return their blood by the inferior vena cava. A large vein, probably the umbilical vein, which has become pervious again, may occasionally be found in the upper margin of the falciform ligament, and when it exists it forms a communication between the portal and general venous system at the umbilicus. The dilatation of veins at this spot, in a few cases of cirrhosis, gives rise to a *caput medusæ* around the

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umbilicus, and forms a communication between the portal and general venous systems. It has been suggested that some of the symptoms of cirrhosis, which are probably toxic, are due to poisons passing easily from the portal to the general circulation by these anastomoses; if so, that is a reason against the operation which will be described later, and which was devised to relieve portal congestion by increasing the anastomoses between the general and portal veins in cirrhosis.

Chronic venous congestion of the stomach and chronic gastritis may often be seen; they are partly directly due to the alcohol taken and partly to portal obstruction. The intestine, too, shows venous congestion and chronic enteritis; this is usually best seen in the duodenum. As Bright pointed out, the large and small intestines may be shortened several feet in cirrhosis; the reason is not clear, but it has been ascribed to the chronic enteritis.

The pancreas may be diseased in those who die with a cirrhotic liver. Out of 6708 post-mortem examinations at Guy's Hospital the pancreas was to the naked eye diseased in 142 instances, and of these 26 were examples of a cirrhotic, congested, or hard pancreas, and in 2 of these there was cirrhosis of the liver. The pancreas, when affected in association with cirrhosis, is rather larger than normal, very tough and hard; under the microscope an excess of fibrous tissue is seen. Many authors consider this condition of pancreas as very common

in cirrhosis, and it has been suggested that backward pressure is not a sufficient explanation, but that here, as in the case of the spleen, the help of a toxin must be invoked to explain the changes.

Causes of Cirrhosis.—The taking of beverages containing alcohol undoubtedly plays, with many patients, an important part in the production of cirrhosis of the liver, for a large number of those who have this disease have partaken excessively of alcoholic beverages; and the size of a hypertrophic cirrhotic liver may become less, and the symptoms may pass away, if the patient turns teetotal early in the course of the disease. In over 60 per cent. of the patients admitted into Guy's Hospital it is easy to obtain a history of alcoholic excess; and when we remember that patients often conceal the fact that they have drunk more alcohol than they should, there can be no doubt that a very large proportion of those who have cirrhosis of the liver have taken alcohol to excess. From this it has been hastily concluded that alcohol is the cause of the cirrhosis, but that it is the sole cause is impossible; for although some children in whom cirrhosis is found have taken alcohol in excess, yet some certainly have not taken any, and in others the amount of alcohol to which the cirrhosis has been ascribed has been altogether too trivial to be guilty; for example, I have known it suggested that the cirrhosis of the liver which

was found in a child was due to the alcohol contained in five minims of tincture of perchloride of iron taken three times a day. Some adults who die of cirrhosis of the liver have not taken alcohol to excess. I made a post-mortem examination upon a friend who died from cirrhosis with ascites, who had all his life been so temperate that he was almost a teetotaller. Then, too, cirrhosis of the liver cannot be produced by the administration of pure alcohol to animals; the liver becomes fatty, but not cirrhotic. Another reason for believing that alcohol is not the sole cause of cirrhosis is that many hard drinkers do not get it; for example, it is not common to see it in women who have peripheral neuritis, and it is excessively rare in asylums and homes for inebriates. Further, it is commoner in some places than others; in London it is common, in Scotland rare, and in Munich alcoholic excess does not often lead to cirrhosis of the liver but frequently to a beer-drinker's heart, but it can hardly be maintained that few people in Scotland or Munich drink to excess. All that can be said is that with some people in some parts of the world over-indulgence in alcohol leads to cirrhosis of the liver and that it does so indirectly, some other associated factor being also operative. It has been suggested that it lowers the resistance of the liver to some other poison, but there is no proof that this is so; the suggestion that it is not the alcohol

but another ingredient of the alcoholic beverages that causes the cirrhosis is hardly likely, for in the first place this other ingredient has not been found, although searched for, and secondly, some of the children and adults who have cirrhosis of the liver have not taken an excess of alcoholic drinks. There is no doubt that alcohol is not solely responsible for cirrhosis, and we are much in the dark as to cause of this condition.

Symptoms.—It is important to remember that the liver may be cirrhotic and yet the patient may have no symptoms of cirrhosis, so that until the doctor, while making a routine examination, finds the liver hard and rough, both he and the patient are unaware that it is diseased. Thus it has been found accidentally in a patient who came up for life insurance. In between a half and a third of all the cases of cirrhosis found in the post-mortem room the patient has died of something else than cirrhosis, and in many of these cases, although he has been under observation in the wards, no symptoms of cirrhosis have been observed. Cirrhosis of the liver producing no symptoms has been found in those dying of tubercle, chronic Bright's disease, and pneumonia, because alcoholic excess is particularly fatal to sufferers from these diseases, but it may be found in those who are apparently healthy but are killed by an accident. Those persons who, if they come under observation, are

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found to have a cirrhotic liver without any symptoms of cirrhosis may frequently, if they give up drinking, continue to live for many years without showing any symptoms. But, on the other hand, a patient with a cirrhotic liver may, at any time, show symptoms of the disease, although the likelihood of this decreases the longer he abstains, but if he lives long enough and continues to drink he certainly will show some or all of them.

As might be expected, the patients are usually over thirty, and as the disease is commonly fatal they are generally under fifty. It is about three times as common with men as with women, why we do not know; but in this connection it is interesting to remember that alcoholic neuritis is commoner in women than in men. It is frequently seen in those whose trade brings them temptations to drink, thus it is not infrequent among publicans and commercial travellers. I should certainly say from my own experience that cirrhosis is a commoner association of alcoholic excess in the lower classes than among those socially above them; this too was the opinion of Sir William Gull and Sir George Burrows.

The patient is usually dyspeptic, but it is difficult to know how far the symptoms of indigestion are due to the cirrhosis and how far to the gastritis set up by the alcohol. The dyspepsia is often severe, there is much dislike of food, especially at breakfast, and the sufferer is often sick on waking. The

bowels are usually constipated. There is often considerable impairment of strength and energy, but here again it is uncertain whether this is owing to the cirrhosis or to the alcohol. There is some pallor, but the skin of the face is commonly reddened; there are dilated venules on the cheeks, and the nose is in a condition of acne rosacea. The tongue is dry and often tremulous. The skin is dry and loose from want of elasticity. It is important to remember that hæmorrhages may occur in parts unconnected with the portal area; thus the nose may bleed and numerous petechiæ may be seen under the skin. Recollection of the fact that cirrhosis is a cause of purpura may save a serious mistake. Small nævi often make their appearance. Various forms of erythema may be seen in association with cirrhosis of the liver; occasionally the erythema on the face is followed by atrophy of the skin, and the condition becomes one of lupus erythematosus. As the case progresses the weakness and languor increase, the muscles waste and become flabby, the bony prominences become visible as the fat disappears, but the wasting is never as striking as in cancer. It is due no doubt, partly at least, to starvation, for the patients often take little food except alcohol, on account of the severe indigestion from which they suffer. The resistance of patients with cirrhosis to bacterial infection is low, and therefore the gums readily

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inflammation, the mouth becomes sore, and decomposition takes place easily in it, with the result that the breath is sometimes very foul. Dyspepsia increases, what food is taken is not properly digested or absorbed, and often the gastritis and enteritis ultimately lead to diarrhoea, which is very difficult to treat. As the general muscular weakness increases so that of the heart, the pulse becomes feeble and rapid, and a certain number of patients suffering from cirrhosis die of gradual cardiac failure. The cardiac sounds are weak and the pulse tension is low. The heart may be fatty, even apart from any hæmorrhage, and there is, if the case is at all advanced, considerable secondary anæmia; how far this is due to the cirrhosis and how far to the wasting and general enfeeblement is doubtful.

Among forty-four cases collected by Carrington, the temperature was irregularly raised in eighteen; but this symptom is often slight, and any continuous rise should make us strongly suspect some complication as tubercle. When in cirrhosis the temperature is raised, apart from any complication, the disease is usually in the active proliferating stage, and hence the liver is enlarged.

We now pass on to certain symptoms which may be more definitely connected with the cirrhotic liver than those just mentioned; such are those local signs in the hepatic region dependent on the alterations of the liver, jaundice, the alterations in

the urine, ascites, the enlargement of the spleen, loss of blood from the gastro-intestinal tract, nervous symptoms, and œdema of the feet.

We have seen from the figures quoted from Dr. Price that at death the liver is oftener found enlarged than diminished in size, and therefore it is not surprising that clinically it is often found enlarged; it may reach down to the umbilicus or even lower, and may be visibly enlarged, the surface may be smooth, but is often finely irregular, and even while the organ is still enlarged well-marked hobnails may be felt. It is important to remember that these are never larger than, at the outside, a small cherry, for remembrance of this fact may help us in deciding whether an enlargement of the liver is due to cirrhosis, syphilis, or cancer. Hobnails are never umbilicated, as cancerous masses may be, and never increase rapidly in size, as cancerous masses may from hæmorrhage into them. Occasionally the cirrhotic process can be made out to be more advanced in one lobe than the other. The liver moves up and down with respiration, the organ feels hard and tough, the edge is regular and hard unless the contraction of the cirrhotic tissue has made considerable progress, when it may be a little irregular; very exceptionally it is thickened from co-existing perihepatitis. Of course, the hepatic dulness is increased, but palpation is a far more reliable means of determining enlargement of the

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liver than percussion. The patient may, if it is large, complain of a dragging discomfort, but severe pain is quite exceptional and usually indicates the super-vention of patchy perihepatitis. The presence of ascitic fluid often makes it difficult to determine whether the liver is enlarged, but then we may sometimes succeed by pressing the hand down suddenly over the liver, "dipping for it," as it is termed.

Whenever at the bedside anything can, in a case of cirrhosis, be learnt about the liver by direct examination of it, we always find it enlarged; for we have no certain means in these cases of detecting atrophy of the liver, because often any diminution of dulness that might be due to shrinking of the liver is obscured by dulness due to ascitic fluid, or coincident emphysema makes estimates of the hepatic dulness uncertain.

Jaundice is present in about a third of the cases of cirrhosis of the liver. It may be transient, and then is occasionally catarrhal, for the duodenal enteritis, which is common in alcoholic subjects, may easily spread up the common bile duct. But it is usually persistent, and is then perhaps due to pressure on the bile ducts by the contracting fibrous tissue. It rarely, if ever, becomes as deep as that seen in carcinoma of the liver. The patient may have the itching of the skin, slow pulse, and other symptoms, due to the circulation of bile in the blood (see chapter on jaundice).

The urine may contain bile if the patient is jaundiced ; but whether it does or does not contain bile it is usually scanty, of high specific gravity, very acid, high-coloured, and deposits a copious precipitate of urates on standing. The amount of urobilin is increased, the urea is diminished, but an excess of uric acid and ammonia are frequently present. Sometimes the dark colour which occurs on adding ferric chloride, and which indicates an excess of diacetic acid in the urine, is especially well marked. Albuminuria is rare, and, when present, suggests that the kidneys are granular.

Ascites, or free fluid, in the peritoneal cavity occurs in about 50 per cent. of all cases of cirrhosis. The abdomen is uniformly distended by the fluid, so that, if there is much of it, both flanks are bulged. The centre of the abdomen is resonant, unless the amount of fluid is very great. The flanks are dull, and each flank becomes resonant when the patient is turned so that the flank is uppermost. A thrill from one flank to the other can usually be obtained. It is said that in ascites the ratio of the distance between the ensiform cartilage and the umbilicus to that between the pubes and umbilicus is unaltered, and that this is a means of distinguishing between ascites and an ovarian tumour ; but the position of the umbilicus is so variable that this means of diagnosis is quite unreliable. Even with the greatest care mistakes

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between ovarian tumours and ascites occur often ; so that it is wise not to tap a supposed ascitic collection in a woman, but rather to let out the fluid through a small opening under an anæsthetic, being prepared to do an ovariectomy if necessary. I have known the taking of this precaution prevent the tapping of an ovarian cyst through the abdominal wall in the case of a woman who had been thought, by every doctor who saw her, to have ascites. If there is a large amount of free fluid in the abdomen the girth of the abdomen may be over fifty inches, the abdominal wall is stretched, the umbilicus is prominent, and lineæ albicantes may form. The muscles of the abdominal wall atrophy, and the veins are consequently very evident. When the quantity of fluid is very great, pressure upwards on the thorax through the diaphragm makes the patient short of breath, the movements of respiration become difficult, and *râles*, indicating congestion and œdema of the bases of the lungs, may occur. I have known such compression of the lung cause a dull note, which led the doctor to think there was fluid in the pleural cavity. This he proceeded to tap low down, and ran his trocar through the pleural cavity and the raised diaphragm, into the abdomen, and so tapped the abdomen through an intercostal space. The patient did well. The heart may be pushed up, and then the impulse is higher than it should be. Sometimes a murmur is heard over the pul-

monary artery, due, it is supposed, to kinking of it, in favour of which view is the fact that the murmur disappears after the abdomen is tapped. The uterus may be depressed.

Pleural effusion often occurs in cases in which there is much ascites ; then it is usually right-sided. We have already alluded to the fact that if ascites is present we may have to "dip" before we can feel the enlarged liver. When we do this we get a sensation of displacement of fluid between our fingers and the enlarged liver, and hence "dipping" is one of the signs of ascites.

The ascitic fluid is usually clear, of a pale green-yellow colour. Its reaction is alkaline, and its specific gravity varies from 1008 to 1015. It contains albumen, a large amount of chlorides, and occasionally a trace of bile, sugar, or urobilin. If peritonitis is present the specific gravity is raised and the albumen is increased. The fact that ascitic fluid contains albumen distinguishes it from hydatid fluid. In very rare instances it has been known to be chylous or to contain blood. The amount of fluid which collects in the abdomen in cases of cirrhosis is often large ; thus, taking ten cases at random, I find the amount removed at tapping to be 12, 23½, 19½, 6½, 8, 11½, 20, 24, 28, 20 pints respectively, and I have seen it collect at the rate of a pint a day. The causation of the ascites and its bearing on prognosis will be discussed presently.

The enlargement of the spleen may be made out by palpation and percussion, but often this is difficult from the presence of ascitic fluid. Very rarely a venous hum may be heard over it, but this is not peculiar to cirrhosis; it may be heard over a spleen enlarged from any cause.

Hæmatemesis occurs in about a quarter of the cases of cirrhosis, and the percentage of men with cirrhosis who have hæmatemesis is higher than that of women. Very few of those who have hæmatemesis die from it. Usually it is profuse, a very large quantity of blood being brought up at once. Indeed, a common experience is for the police to bring to the hospital a man who has been picked up in the street in the act of vomiting a large amount of blood, and on admission it is found that the cause of this is cirrhosis of the liver. Not uncommonly it is the first symptom of cirrhosis, and it is usually an early symptom. If hæmatemesis is frequently repeated the patient has usually not got cirrhosis, but gastric ulcer or gastrostaxis. The vomited blood in cirrhosis is usually dark. The patient suffers from collapse, the degree of this depending upon the amount of blood lost. Usually some blood passes the pylorus, and hence *melæna* is present. The most important cause for hæmatemesis when associated with cirrhosis is rupture or ulceration of a varicose vein at the lower end of the œsophagus, for, as has already been pointed out, the

veins in this situation are often dilated in cases of cirrhosis of the liver. Rarer causes for hæmatemesis in cirrhosis are the gastritis which sometimes accompanies it, minute erosions of the gastric mucous membrane, and in extremely rare instances a gastric or duodenal ulcer. Although usually melæna is due to bleeding from the stomach, it may be due to hæmorrhage from any part of the gastro-intestinal tract. It is often stated that piles are common in cases of cirrhosis, and we should have expected them to be so, but they certainly are not. It is said that, when present, bleeding from them is beneficial, as it helps to empty the already overfull portal venous area. On p. 134 it was explained that occasionally dilated veins might be seen around the umbilicus. Students often exaggerate the frequency of this. They are seldom seen, and when present are small.

Nervous symptoms are common in cases of cirrhosis; some, *e.g.* those of delirium tremens, are directly due to the alcoholic drink; others, *e.g.* coma, may be due to the jaundice, but nervous symptoms, especially coma, are often present, and are frequently fatal in those who have cirrhosis but have not recently taken alcohol and among those who are not jaundiced. Delirium—not delirium tremens—is sometimes seen and may continue till death, but it is rarer than coma, and convulsions are rarer still. It is, as Mott points out, remarkable that although

alcoholics are largely represented in asylums, cirrhosis of the liver is not common there.

The swelling of the lower extremities which supervenes in some cases of cirrhosis is often set down to the pressure of the ascitic fluid on the vena cava ; but this certainly cannot always be the cause, for it is not infrequent to find that the swelling of the feet and ankles appears before any distension of the abdomen, and it may even be considerable in a case in which post-mortem examination shows only a small amount of fluid in the abdomen. It may, too, occur when the liver is of normal weight, so that pressure of an enlarged liver on the vena cava is not the cause, and it may be seen in those in whom the anæmia is not severe. The cause of it will be discussed presently ; here all we need say is that it usually indicates that the patient is suffering severely from his cirrhosis.

Explanation of the Symptoms.—The explanation of some of the symptoms, *e.g.* the hæmatemesis and the jaundice is obvious, and has been alluded to in the description of them. Cirrhosis of the liver has been regarded solely as a local disease of the liver, but that view makes it difficult to understand all the symptoms. For example, it has been stated that the pressure of the contracting fibrous tissue on the branches of the portal vein in the liver is the cause of the ascites, but a moment's thought will show that this cannot be so ; for I have been able,

from a study of the history of many cases, to make out that the fluid in the abdomen frequently collects, almost suddenly sometimes, at the rate of a pint a day, which would be very difficult to understand if we believed that the slowly forming fibrous tissue, by its compression of the portal veins within the liver, is the sole cause of the ascites. Again, a high degree of cirrhosis may often be found at the post-mortem in cases in which the disease was unsuspected during life, and in which no ascitic fluid existed in the abdomen. Then too the presence of ascitic fluid in the abdomen bears no relation to the size of the liver; but if it followed from pressure of the fibrous tissue it ought to be most evident when the liver was atrophic, nor is ascitic fluid most often found in those cases in which the various anastomes between the portal and general venous system are dilated, although we may assume that in them the portal pressure is high; indeed, it is often found in cases in which these veins are not dilated, and there has been no hæmatemesis. It has been suggested that the cause of the ascites is thrombosis of the portal vein itself or its minute branches in the liver, but this is very rare, and ascites is common. Herrick states that there is no obstruction to the portal vessels from fibrosis in the large cirrhotic liver, and, even if there were, Dr. Bolton showed that slight constriction of the portal vein does not produce ascites; rather

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greater constriction produced some slight ascites, but the fluid was soon absorbed. Thus we see that increased portal pressure cannot be the sole cause of ascites.

Probably the correct view is that which I urged fifteen years ago, and which has been adopted by others, namely, that we ought to take a wider view of cirrhosis of the liver than is implied in the statement that it is a local disease of the liver, and to believe that as a result of the damage to the liver, or possibly from some other unknown cause, certain poisons circulate in the blood. It may be that these get there because poisons, which are formed possibly in the alimentary canal, are in health destroyed by the liver, and, when that organ is cirrhotic, pass into the circulation; or it may be that in cirrhosis the poisons are formed in the liver, or that some normal internal secretion of the liver is prevented; but anyhow we must, I think, assume that in many cases of cirrhosis a poison does circulate in the blood, and that it has a lymphagogue action. This will explain why swelling of the feet frequently occurs, even when there is no pressure on the vena cava; it will explain the occurrence of ascites, although no doubt this is sometimes helped by pressure on the portal vein; it will explain the fact that pleural effusion is not infrequent.

Probably, too, other symptoms are due to this poison, such as the coma, convulsions and delirium,

which may occur quite apart from jaundice. Then, too, although want of food and dyspepsia may help to explain the wasting weakness and anæmia, yet sometimes these are so evident as to suggest that they are due to some poison circulating in the blood, and the occasional rise in temperature may be due to the same cause; indeed, it is difficult to understand, unless we invoke the aid of such a poison, why cirrhosis should be so fatal. Probably the mere cirrhosis of the liver does not itself matter much so long as the poison does not get into the circulation; but a person with a cirrhotic liver is at any moment liable to this, and then he quickly becomes dangerously ill—a view supported by the fact, which will be evident when we discuss the prognosis, that many cases die quickly, soon after serious symptoms, especially ascites, swelling of the feet, and nervous symptoms arise. On this view a person with a cirrhotic liver is comparable to one with granular kidneys, for such a one may apparently be in fair health and suddenly serious symptoms of uræmia arise.

It has been suggested that the ascites is due to concomitant inflammation of the peritoneum or to cardiac failure; but not only is there rarely any evidence for either of these views, but neither would explain the other symptoms, such as swelling of the feet and nervous disorders, which are well accounted for by the view just enunciated.

Associated Conditions.—Inasmuch as those who drink more alcohol than they should are prone to tuberculosis, it is not surprising that patients with cirrhosis often suffer from various forms of tubercle. Newton Pitt's figures show that alcoholic cirrhosis in patients under forty is accompanied by tuberculosis in two-thirds of the cases. Usually there are no signs of it during life, and the patients die more often from their cirrhosis than from their tubercle. The most frequent seat of the tubercular disease is in the lungs, next the peritoneum; and thus it comes about that sometimes the supervention of ascites has not the serious significance that it has if it is directly due to cirrhosis, for, among some of those who have cirrhosis, it is due to tubercular peritonitis. It is stated, and may well be believed, that tubercular disease is not specially common in the rare cases in which the cirrhosis is non-alcoholic. A tubercular cirrhosis—that is to say, a cirrhosis due to actual tubercular disease, has been described, but does not exist.

There is a form of chronic peritonitis, usually called simple chronic peritonitis, which is not tubercular, and which often accompanies cirrhosis of the liver, probably in between 10 and 20 per cent. of the cases; it is therefore the explanation of some of the cases of ascites occurring in those who have cirrhosis.

Fatty degeneration of the heart may occur in

association with cirrhosis, and is due, no doubt, to the over-indulgence in alcohol.

The kidneys are usually healthy, but sometimes they are enlarged from the excessive drinking and consequent excessive secretion of urine; sometimes there is some fatty change, probably due to the alcohol, and sometimes they are congested from cardiac failure. There has been much dispute as to whether the kidneys are frequently granular. Inasmuch as granular kidneys are not infrequent in people past middle life, and cirrhosis, too, is mostly seen in these, we should expect granular kidneys and cirrhosis of the liver to overlap; this they certainly do, but whether more often than chance would lead us to expect has not yet been determined.

Arterio-sclerosis and atheroma are present in those who have cirrhosis, but it must be remembered that both are often seen at about the same age as cirrhosis. When these are associated with cirrhosis, probably the arterial changes are due to the alcohol directly and not to the cirrhosis.

The relations between carcinoma and cirrhosis are discussed in the chapter on "Carcinoma of the Liver."

It has already been mentioned that pleural effusion is not infrequently seen in association with cirrhosis. It is usually right-sided and may sometimes be tubercular; but certainly sometimes it is not, and then is perhaps due to the circulation of

the lymphagogue poison, which has been suggested. The effects on the lungs and heart of extreme ascites were described on p. 144.

Peripheral neuritis is not, I think, as often associated with cirrhosis as might be expected.

Diagnosis.—The difficulties of diagnosis usually fall into one of two classes, either what is the cause of an enlargement of the liver, or what is the cause of an ascites. With regard to the enlargement of the liver, we have to remember that the liver may appear enlarged when in reality it is displaced downwards (see p. 6), or apparent enlargement may be due to causes outside the liver, *e.g.* tumours of other organs near it. These are mentioned in describing the diagnosis of cancer of the liver (see p. 224). The most frequent difficulty is to decide whether an enlarged liver is the seat of cirrhosis or cancer, but this is discussed on p. 226. Syphilis of the liver does not cause much confusion, for it is very rare at the bedside. The irregularities in the organ are much larger than the hobnails of cirrhosis, and the patient with a syphilitic liver is rarely jaundiced and hardly ever has ascites. I have known a difficulty to arise between the cirrhosis of children and congenital syphilis of the liver.

Obstruction of the common bile duct leads to a large smooth liver. When this is due to a gall-stone there is usually deeper jaundice than in cir-

rhosis, but no ascites; the stools are quite white, which is very unusual in cirrhosis, and there is commonly a history of gall-stones. When the obstruction is due to malignant disease of the head of the pancreas, there are often secondary nodules to be felt in the liver; the stools are white, the jaundice becomes very deep, and there is no ascites.

If the patient has had malaria, he may have taken too much alcohol and so have an alcoholic cirrhosis. There probably is no such malady as malarial cirrhosis. The liver may be enlarged as a result of malaria, but then it is rarely as much enlarged as in cirrhosis; it is quite smooth, there is no ascites, and the spleen is always very large. Although the liver may be enlarged in many other conditions, they hardly ever give rise to difficulties of diagnosis.

When a patient has ascites the cause of it is often by no means clear, but the first thing we have to determine is whether the enlargement of the abdomen is due to ascites or an ovarian tumour. Usually the tumour can be felt; it is local in one part of the abdomen, it rises out of the pelvis, and is more to one side than another; but other points of distinction have been given when discussing the diagnosis of ascites (see p. 143). It should be added that a large ovarian tumour often drags up the roof of the vagina, so that it may be difficult

to reach this with the finger, but a large collection of ascitic fluid may depress it.

The determination as to whether a collection of ascitic fluid is due to cirrhosis of the liver is hampered by the fact that the very presence of ascites makes it difficult to feel the liver. When heart disease or renal disease is the cause of ascites, mistakes are rare; the usual difficulty is to tell whether the ascites is due to cirrhosis or some form of chronic peritonitis. If this is tubercular the fact that the patient is most often a child, the feeling of irregular masses of thickened tuberculous peritoneum, and the finding that the fluid is loculated may all be of great help. Continued fever is much in favour of tubercular peritonitis; jaundice never occurs with this disease. The history, other evidence of tubercle or of alcoholic excess, and the nervous symptoms may all be of aid, but it must be remembered that cirrhosis and tubercular peritonitis not infrequently co-exist.

Malignant disease of the peritoneum is rare, but is usually distinguished by feeling masses of malignant disease in the abdomen.

A common difficulty is to tell whether ascites is due to cirrhosis or to simple chronic peritonitis. The most important point of distinction is that, if the ascites is due to cirrhosis of the liver, the patient is usually dead soon after its appearance, and rarely lives long enough to survive a second tapping, as will be shown

presently when we discuss the prognostic value of ascites. On the other hand, sufferers from chronic peritonitis often survive many tapplings; it may be even thirty. Chronic peritonitis does not give rise to jaundice, and if the liver is affected by it we have the condition known as perihepatitis (see p. 202); the liver then is smooth, with a thick edge, and is thus in striking contrast to a cirrhotic liver. The ascites of chronic peritonitis is often associated with a thick puckered omentum lying as a tumour transversely across the abdomen, and other thickenings of the peritoneum may be felt; if so the fluid is very liable to be loculated, and the inflamed mesentery, becoming contracted, drags the intestines back, so that with a comparatively small amount of fluid the abdomen is dull all over; but it must be remembered that during life it may be impossible to feel the thickenings or to recognise that the fluid is loculated, and further that chronic peritonitis is often associated with cirrhosis.

If the ascites is associated with malignant disease of the liver, the diagnosis becomes one between that disease and cirrhosis. That is discussed on p. 226.

Prognosis.—About half the cases in which cirrhosis of the liver is found in the post-mortem room die from some other disease, and the majority of these patients have not shown any symptoms of cirrhosis. It is not unreasonable to suppose of some of them that if they had not had the disease that

killed them, and had given up indulgence in alcohol, they would, as years went on, have shown no symptoms of cirrhosis. I have known a man, in whom a hard cirrhotic liver was detected in the course of a routine examination, give up alcohol and live in excellent health for many years (see p. 138). It has been suggested that the reason why these patients have shown no symptoms is that the compensatory hyperplasia of the hepatic cells has been sufficient.

When the patient begins to show symptoms the outlook becomes very serious, but even then, if he becomes a teetotaller, he may occasionally live for years. A few instances have been published in which a patient has had ascites and other symptoms of cirrhosis, has become a total abstainer, has lived many years before he died of some other disease, and then after death cirrhosis of the liver has been found. In some, at least, of these cases the ascites may have been due to some other cause than the cirrhosis.

Much discussion has taken place about the prognostic value of ascites, but there appears no doubt that, when it is not due to simple chronic peritonitis or tubercular peritonitis, its supervention in cirrhosis means that the patient will die within two or three months. To investigate this point I took a number of fatal cases from the records of Guy's Hospital. In 10 cases ascites was present, but the patient was not tapped; but, as far as could be ascertained from the history, the time from first noticing that

the abdomen began to enlarge till death was on the average only two months, and the same was true of 12 that were tapped. In 4 cases death occurred within a month of the commencement of the enlargement of the abdomen, and in only 2 of those that were tapped was life prolonged beyond three months. These cases strongly support the statement just made as to the seriousness of the onset of ascites. Dr. Campbell Thomson studied 18 cases from the Middlesex Hospital, and concluded that they "in every respect support Dr. Hale White's conclusions." His cases, like mine, when they were tapped died within a few weeks of tapping, and some cases recently published by Dr. Ramsbottom also show the extreme seriousness of the onset of ascites, for after most careful consideration he concludes that "in the great majority of cases of alcoholic cirrhosis of the liver, in which the peritoneum is not thickened as shown post-mortem, ascites is a terminal event." Austin Flint, Watson, Niemeyer, Fagge, Murchison, Wickham Legg, and many others have also emphasised the same point. Although, if the amount of fluid present is great, it may impede respiration and disturb the action of the heart, it is very unlikely that the mere presence of ascitic fluid causes death, for many patients die when the amount is still small, its removal does not ward off the fatal end, and in patients in whom ascites is due to some other cause, several hundred

pints may be poured out before death closes the scene. The ascites of cirrhosis is to be taken as indicative that the patient is near his end. A post-mortem examination upon any patient having cirrhosis, who has been tapped often, will show that, although he has cirrhosis, he also has some form of chronic peritonitis; and, as when no peritonitis is present cirrhosis is so rapidly fatal after the onset of ascites, it seems fair to assume that the long-lasting ascites was due to the chronic peritonitis rather than the cirrhosis.

The appearance of swelling of the feet in a person having cirrhosis of his liver is a grave event, for life is rarely prolonged more than a few weeks after it becomes evident. The same is true of multiple hæmorrhages in the skin or mouth.

The onset of nervous symptoms is likewise very serious, and patients in whom well-marked coma has supervened very rarely recover. We have already seen that ascites, swelling of the feet, hæmorrhages, and nervous symptoms are all probably toxæmic. A patient with cirrhosis may have the disease for some time without any symptoms, but he is liable at any time to the supervention of ascites, swelling of the feet, hæmorrhages, or nervous symptoms, and any or all of these mean that the end is near. So striking is this that it has been suggested that the removal of the ascitic fluid is a cause of the coma. This is unlikely, but at any

rate the removal of the fluid does not usually benefit the patient.

Hæmatemesis is not nearly so serious as general hæmorrhages, for it is not toxæmic, but is due to raised portal pressure. It is often an early symptom and is rarely severe enough to be fatal. Indeed, it is often beneficial, for it calls attention to the fact that the patient has cirrhosis.

The older the patient the more likely he is to die; this probably because he has been drinking longer than the younger man. Inasmuch as the liver is large in the earlier stages, it is not surprising to learn that most of the cases that have recovered have had large livers. Patients who are thin and anæmic as a rule do badly. The onset of any microbic infection, such as pneumonia, tubercle, or abscess, renders the prognosis very grave.

Treatment.—If the toxic symptoms have not shown themselves, and the cirrhosis is only recognisable by the presence of an enlarged irregular liver with some indigestion and perhaps a single hæmatemesis, the patient stands a fair chance of recovery if he becomes a teetotaller, takes plain, simple food, and keeps the bowels open regularly, preferably with a little calomel. If jaundice has come on, treatment is not so likely to be efficacious, but still the outlook is by no means hopeless. The doctor must be very strict that the patient becomes an absolute teetotaller. If permission is given for

a little alcohol, he is very likely sooner or later to exceed, and even small amounts may be harmful to a man whose liver is already cirrhotic. Of simple foods milk is the best, and, if he can manage it, the patient should for a time take three or four pints of milk a day and nothing else, then bread and butter, milk puddings, and infants' foods may be given for a few weeks, and then eggs, and finally a plain but more varied diet. Going to some place, such as Harrogate, Homburg, Carlsbad, or Marienbad, where the patient will be carefully dieted and drink aperient waters, often does good. Any dyspepsia that is present should be carefully treated.

If hæmatemesis comes on the patient should be kept quiet in bed, and for three or four days he should be fed by the rectum. Perhaps the best nutrient enema consists of the yolk of two eggs, 0.5 grm. (7.5 gr.) of common salt, 50 grms. (1.75 oz.) of pure dextrose, and 300 c.c. (10 fl. oz.) of milk. As the hæmatemesis from cirrhosis usually soon stops, it will be enough to give one of these three times a day for two or three days. If collapse is extreme, it may be necessary to give a saline injection by the rectum. This should not be done unless absolutely necessary, for it raises the blood pressure and so may induce fresh bleeding. Do not allow ice to be sucked; it only increases the peristaltic movements of the stomach, and so leads to fresh hæmorrhage. A subcutaneous injection of morphine

is very beneficial, as it keeps the patient quiet and calm. For the thirst the patient may rinse out his mouth with warm water. Drachm doses of calcium chloride may be dissolved in a little water, and given by the rectum twice in the twenty-four hours to increase the coagulability of the blood. Usually before giving either this or the nutrient enema it will be necessary to wash the rectum out with soap and water. Ergot has been much used, but it raises the blood pressure and so does more harm than good. Adrenalin should never be injected subcutaneously, for given thus it raises the blood pressure, but it may be given by the mouth, for it is not absorbed from the stomach, and may perhaps chance to come in contact with the source of the bleeding and so check it. Twenty minims of the 1 in 1000 solution is a suitable dose. Half a drachm of the liquor of perchloride of iron with a drachm of glycerine, to render it easier to swallow, has often been given by the mouth with the object of acting as a local styptic on the stomach. The disadvantage of it is that it is so nasty it may make the patient sick. Preparations containing tannic acid have been given in the same way, but drugs having for their object the arrest of hæmatemesis are hardly required in cases of cirrhosis, for a moderate loss of blood is often beneficial, and the amount lost is rarely sufficient to be dangerous. On the third day after the cessation of the hæmorrhage a little peptonised

milk may be ordered, and gradually more and more may be taken. For some time the food should be very plain, and the sufferer should, of course, become a teetotaler.

If coma and other nervous symptoms are present, an aperient should be given in sufficient dose to secure that the bowels are open loosely, and the patient should be put in a hot bath or given a hot air bath to make him sweat, for by these means the excretion of the poison will be helped. Saline transfusion is sometimes of great use, no doubt because the circulation of a large quantity of fluid dilutes the poison and aids its excretion. If the reaction for diacetic acid is present in the urine, two drachms to the pint of bicarbonate of soda should be added to the water used for transfusion.

Much discussion has taken place about the treatment of ascites; there is no doubt that purgatives and diuretics will often decrease the amount very considerably. Of the diuretics copaiba resin is the best. Fifteen grains may be given in a cachet, or this dose may be stirred with 20 minims of alcohol (90 per cent.), and thoroughly stirred again with a little thick mixture of compound tragacanth powder and water, syrup of ginger may be added, and the whole made up with water to 1 fluid oz. The great objection to copaiba resin is that it is very nasty, but it is so often efficacious that it should be

given a trial. The well-known pill containing a grain each of powdered digitalis leaves, squill and blue pill may be tried if copaiba fails. There are, however, many cases that drugs do not influence, and with regard to them two points have to be discussed, viz. the propriety of letting out the fluid and the propriety of what is known as the Talma-Morison operation.

Unless the quantity of fluid is so great that it hampers the movements of respiration or pushes the heart out of place, there is nothing to be gained by letting it out. Indeed, patients so frequently die if they are ill enough for the amount of fluid to be considerable, that, as Fagge said, the removal of the fluid, whether by drugs or mechanically, seems to hasten the end. This in all probability is not so. The patient is so ill that he would have soon died anyhow. A Southey's tube and fine rubber tubing is often used; it is best put into the side of the abdomen at the lower part, provided of course that the abdomen is dull there. The trocar must be plunged in only where a dull note is obtained. Better still is an ordinary fine trocar and canula, with rubber tubing attached to the canula, for a Southey's tube is so small that it or the tube connected with it often gets clogged. In either case the tubing is so arranged that the fluid runs into a pail at the side of the bed. Of course, the canula should be boiled and all aseptic

precautions taken. After the fluid has been let out the opportunity should be taken to feel the liver, for it is then usually very easy to determine its size and to ascertain whether it has any irregularities on it. A binder should be put on the abdomen, which should be gradually compressed with it while the fluid flows, and the binder should be fixed with safety-pins and left on for a few days. A collodion dressing is put on the site of puncture. Sometimes the fluid continues to drain away through the puncture for a few days after the canula is removed. There is no harm in this, and it may soak into an antiseptic dressing. Indeed, many attempts have been made to arrange that the fluid shall run away continuously through a hole in the abdominal wall, but none of them have been successful.

A surgical method of treatment, based on the assumption that the ascites of cirrhosis was due to portal obstruction, and therefore might be relieved by increasing the anastomoses between the portal and general venous systems, was devised independently by Talma and Morison, and hence it is often known as the Talma-Morison operation. Morison wrote his original paper in connection with Drummond. This is not the place to describe the various surgical modifications of the operation. They have been discussed by Sinclair White, and in his paper, and in one by Lawrence Jones, full references to the literature will be found.

Lawrence Jones considers Morison's original operation the best. In it, after the abdomen is opened by an incision six inches long, the peritoneum, especially that covering the liver and spleen, are rubbed with gauze so as to irritate them; the omentum, similarly treated, is spread out and sutured to the parietal peritoneum, over an area as wide as possible, and the incision is closed. A glass tube is left in the abdominal cavity at its lower part, so that the ascitic fluid, which continues to be poured out for some weeks after the operation, may drain away. The abdomen is tightly strapped. It is excessively difficult to arrive at an opinion as to whether the operation does much good, but at least a third of those upon whom the operation has been performed die within a month, and as there is a great likelihood that successful, rather than unsuccessful, cases will be recorded, it must be that in many cases the end has been hastened by the operation. About a third of the cases are stated to be cured, but in considering them it must be remembered that some of the cases recorded as cirrhosis of the liver were not uncomplicated examples of this disease; they were probably instances of slight cirrhosis with some other cause for the ascites. Again, often a case has been considered cured although we only know of its after-history for a few months. Further, the very word "cure" is inappropriate when applied to

a liver so far cirrhotic that ascites supervenes, for such a liver can hardly become healthy again. Then, too, it must sometimes be difficult, at an operation, to give an opinion as to whether a liver is cirrhotic or not, and in some of the cases considered as cured, probably the patient had no cirrhosis at all, but the ascites was due to other causes, *e.g.* tubercular peritonitis, which admit of cure by tapping, without the Talma-Morison operation; and finally, even if some cases of cirrhosis have benefited, we must set against the prolongation of their life the shortened life of those in whom death followed the operation.

It must not be forgotten that there are many reasons why we should not expect the operation to do much good, for we have seen there are strong arguments in favour of the view that ascites is not principally due to increased pressure in the portal vein, but is dependent chiefly upon a lymphagogue toxin acting on the peritoneum in the same way as it causes swelling of the feet of those who suffer from cirrhosis; and further if, as is quite possible, the portal blood of patients with cirrhosis contains toxins, the increase of the anastomoses between the general and portal venous system may increase the toxæmia, just as when in dogs the portal vein is connected with the inferior cava, (Eck's fistula) severe nervous symptoms follow. Putting together my own experience and infor-

mation gained by reading, I think there is no doubt that the operation has often been performed in unsuitable cases. I have rarely seen a case in which there was the slightest probability of an operation doing good, for, as I have already urged, the very fact that ascites has supervened in uncomplicated cirrhosis, means that the patient is near his end. The whole question has been recently very fully discussed by Lawrence Jones. He, too, finds the mortality very high, and his final conclusion is that the majority of examples of ascites with cirrhosis do not seem suitable for this form of treatment. He believes that the cases of cirrhosis which are suitable are those which have survived one or two tapplings, who present obstructive rather than toxic symptoms, and whose general condition is fairly good; but, as he truly says, such are not common.

Quite recently Dr. Essex Wynter and Mr. Sampson Handley have drawn the peritoneum through the femoral ring and then split it, hoping that the ascitic fluid will pass into the tissues of the thigh and be absorbed from there. In their case the fluid had not, ten weeks after, collected again in the abdomen.

Cirrhosis in Children.—We occasionally find children affected with cirrhosis of the liver quite indistinguishable from that just described—that is to say, the fibrous tissue of the portal canals grows in among the hepatic cells, producing first a multi-

lobular, then a unilobular, and then an intralobular cirrhosis. The liver becomes hard and hobnailed. This variety of cirrhosis is of great interest, for although in many of the cases the child has had an excessive amount of alcohol—I have seen a boy with cirrhosis of the liver a confirmed drunkard at the age of eleven—yet in others there is no doubt that the cirrhosis has taken place in children who have not taken alcohol. We have already seen that it is impossible to believe that alcohol is the sole direct cause of cirrhosis of the liver, and the fact that it may be found in children who have not taken any alcohol is strong evidence in support of this belief. We are completely ignorant of the cause of this variety of cirrhosis in these patients, whether children or adults, who have not taken alcohol. The symptoms, outlook, and treatment are the same in both children and adults, except that, as in children, the healthy liver is proportionately larger than that of adults, so the cirrhotic liver is especially large; in children the spleen distends more readily than in adults, so the cirrhotic spleen is unusually large; and as the temperature of the body is more unstable in children than in adults, so pyrexia is more frequent and better marked in children than in adults who suffer from hepatic cirrhosis. Forgetfulness of this has led to errors of diagnosis.

There is an excessively rare disease, the main symptoms of which are nervous, namely, loss of

power, muscular contractions, and mental weakness, associated with some fever and great wasting. After death cirrhosis of the liver is found. This disease occurs in children, and often several members of one family are affected.

Hanot's Cirrhosis.—This is a rare disease, often called hypertrophic biliary cirrhosis—a very bad name which had better be discarded, for, as we have already seen, the liver is frequently enlarged in ordinary cirrhosis; and it is a pure assumption that the cirrhosis in Hanot's cirrhosis is biliary, it being meant by that word that the cirrhosis is due to chronic inflammation of the biliary passages set up by inflammation spreading up from the duodenum.

The term hypertrophic biliary cirrhosis has been so often used, that before considering Hanot's cirrhosis it will be well to clear away a few misconceptions. Some, especially French writers, have maintained that the cases of ordinary cirrhosis in which the liver is large are examples of a distinct disease, which has been called hypertrophic biliary cirrhosis, but there is no doubt that the cases so described are really examples of the early stage of ordinary cirrhosis. It is said that in this so-called hypertrophic biliary cirrhosis the liver is not hobnailed, but in atrophic cirrhosis it is; but we often see a hypertrophic cirrhotic liver in which the hobnails are well marked. I have seen them

very distinct in livers weighing between 70 and 80 ounces. Of course, as a rule, the smaller the liver the more marked is the hobnailed appearance, for contraction of the fibrous tissue means that the liver will become small and the hobnails well marked. Next, it is stated that in this so-called hypertrophic biliary cirrhosis the increase of fibrous tissue begins round the biliary passages, surrounds single lobules, and goes between the cells of each lobule, and hence is monolobular, and intercellular, and not multilobular, and further that the rows of liver cells, which were formerly considered as new bile ducts (see p. 130), are only seen in this form of cirrhosis. Leaving aside for the moment the question whether this histological description is correct for the few cases of large liver to which the name Hanot's cirrhosis should be applied, in cases of ordinary cirrhosis in which the liver is large, the new fibrous tissue begins in association with the portal vein quite as much as with the bile ducts, and it is multilobular as well as monolobular and intralobular, and rows of new liver cells can be seen; indeed, the prevalent opinion in this country is that no histological distinction can be drawn between the so-called hypertrophic biliary cirrhosis and ordinary cirrhosis; certainly we should hardly expect a precise difference of distribution of fibrous tissue, and in many cases we see multilobular, perilobular, and intralobular cirrhosis in the same liver. Further, it is

said that when the liver is large no ascites is present, but this is not so. I have often seen a large cirrhotic liver with ascites. In one case in which at death the liver weighed 74 ounces, 36 pints of fluid were withdrawn from the abdomen three weeks before death. We have seen that ascites is often a terminal event, and therefore naturally it will frequently be seen with a small atrophic liver, for we have already learnt that this is an advanced stage of the disease, and that the ordinary large cirrhotic liver shrinks and becomes small if the patient lives. Again, it is maintained that when the liver is large the jaundice is deep and long-standing, but, as a matter of fact, the jaundice is often absent when the liver is enlarged; nor is it true that nervous symptoms are more likely to be present when the liver is large, and we have seen that there is no relationship between the size of the spleen and that of the liver, although it has been said that the spleen is larger when the liver is large than when it is small. It has been stated further, that when the liver is large the patient has not taken an excess of alcohol, but that certainly is not so. Over and over again in hospital work we come across patients who have drunk hard and have large cirrhotic livers; indeed, Rolleston found that a history of alcoholic intemperance was slightly more common in those in whom the liver was large than among those in whom it

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was small. It has been stated that albuminuria is less often associated with a large liver than with a small, but this is doubtful, and, even if true, it would not be surprising; for if, as I believe, the large cirrhotic livers later become small, those patients with small livers will be older than those with large, and hence will be more liable to granular kidneys. This consideration also explains the supposed distinction that those with large cirrhotic livers are usually younger than those with small; and again, the statement that persons with hypertrophic cirrhosis live longer than those with atrophic is of no value as a distinction, as it is obvious it must be so, for many of the hypertrophic cases will live on through this stage; but when a man gets to the atrophic stage he has no further to go. Lastly, it has been stated that the proportion of males is higher in hypertrophic than atrophic cirrhosis and that pyrexia is commoner in the hypertrophic form, but neither of these distinctions hold.

These points have been considered in this detail, because if the liver is large the disease is often labelled hypertrophic cirrhosis or hypertrophic biliary cirrhosis, as though this was a distinct disease; but it cannot be too strongly insisted that there is no such distinction between the ordinary atrophic and hypertrophic cirrhotic liver. With the exception of one or two excessively rare diseases now to be described, all that can be clinically called cirrhosis of the liver

is one and the same disease; any variations in the size of the liver or the symptoms depend upon the stage of the disease. I used the word clinically so as to exclude slight increase of fibrous tissue, such, for example, as that seen in a nutmeg liver, which cannot be detected except by histological examination, and which is of no clinical interest. A cirrhotic liver, which is an example of the common disease cirrhosis, is easily recognised without the aid of a microscope. Further, it must be remembered that as cirrhosis of the liver is a common disease in some countries, so any one working there will, before he has been engaged in clinical work many years, have seen many cases and therefore several combinations of symptoms, so that he may well have seen patients in whom the liver is large and not hobnailed, the jaundice is deep and there is no ascites; but he would no more be justified in separating such patients as examples of a separate disease than would any one with much experience of typhoid fever be justified in separating those cases which had abundant diarrhoea and few spots as examples of a disease distinct from other cases of typhoid. It is clear from this discussion that it will be wise to discard altogether the phrase "hypertrophic biliary cirrhosis," and only to use the term hypertrophic cirrhosis as indicating that the disease cirrhosis of the liver is in such a stage that the liver is large.

We now return to the description of that rare

disease best known as Hanot's cirrhosis; but it, like the large stage of an ordinary cirrhotic liver, has been called both hypertrophic cirrhosis and hypertrophic biliary cirrhosis, but for the sake of clearness neither of these names will be used.

Hanot's cirrhosis is uncommon. It is named after Hanot, not because he first described it, but because his thesis first directed attention to it. Very many of the cases occur in children, but not in infants. A few of the sufferers attain the age of thirty years. It is commoner in males than females. The characteristic features of it are that the disease lasts many years, the liver is firm, enlarged and smooth, long-standing jaundice is present, the spleen is very much enlarged, so that Hanot's cirrhosis has been called spleno-megalic cirrhosis; but this should certainly never be done, the term should be reserved for Banti's disease (see p. 181).

The considerable enlargement of the liver is uniform, and weights of over 100 oz. have been recorded, but it is rarely more than double its normal weight. It must be remembered that often the patients are children of stunted growth, so that a liver may be especially large for the particular patient; for example, in a case which was published by Dr. F. Taylor, the child, who was fourteen years old, was only very slightly over four feet high, but his liver weighed 40 oz. and was felt $3\frac{1}{2}$ inches below the ribs. In very long-standing cases the surface

of the liver may, towards the end of life, be a little uneven from contraction of the new fibrous tissue, but it never has the hobnailed appearance of ordinary cirrhosis. Histologically the new fibrous tissue is much more delicate than that of ordinary cirrhosis. It is at first monolobular, later it becomes intra-lobular, and, later still, a little pericellular cirrhosis develops. The wrongly called new bile ducts (see p. 130) are very numerous. The liver is stained a deep yellow.

The spleen is proportionately much more enlarged than the liver; in Dr. Taylor's case, just mentioned, it weighed $87\frac{1}{2}$ oz., but this was exceptional. Still it often reaches 40 oz. There may be a little perisplenitis. Histologically the spleen tissue appears normal, except that the vessels are very full of blood, and in long-lasting cases there is some increase of fibrous tissue. There must, therefore, be great hypertrophy of the splenic pulp.

Jaundice is an early symptom, and it lasts till the end, so it may be present for many years. The intensity of it changes very slowly, but gradually it becomes deep and may in the end be dark green; but so slow is the deepening that the patient may die before it is very dark. From time to time the patient has periods during which he feels ill and his temperature is raised; then the jaundice deepens a little. As the jaundice is so long-lasting, xanthelasma (see p. 34) is sometimes seen.

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Patients afflicted with Hanot's cirrhosis live a long while. Dr. Taylor's patient, whom I often saw, had it six years before he died. For the first few years the subjects of it remain well in spite of the jaundice. A boy under my care, probably an example of this disease, used, although deeply jaundiced, to run about and play as though nothing was the matter with him; his tongue was clean, his appetite good, and he had not that dislike for fats so often seen in those who are jaundiced. As mentioned just now, the sufferers are liable to attacks of malaise and a slight rise of temperature, often associated with pain in the abdomen; these gradually become more frequent, the patient begins to waste and lose strength, the jaundice deepens, and he dies, apparently from poisoning by bile, for he has the drowsiness, occasionally accompanied by delirium, to which the term *cholæmia* is applied; but he may die from some terminal infection.

Ascites is rare and usually comes on late in the disease, and therefore is of serious significance. Dr. Taylor's patient died six weeks after ascites was first noticed. Many of the patients die without any ascites being found, even in the post-mortem room.

A very strange symptom is that in several of the cases the fingers have become clubbed; this is more likely to occur in children than adults. The bones are unaffected; the swelling is entirely in the

soft parts. The clubbing is exactly like that seen in chronic pulmonary phthisis. The cause is quite unknown; indeed, we do not know why the fingers, and sometimes the toes, should be clubbed in any of the diseases in which this appearance is seen.

If the disease begins before growth ceases the patients are often stunted. Dr. Taylor's patient was only 4 st. 4 lbs. at the age of twelve, and only 4 ft. 1 in. high at the age of thirteen. The average weight of males at twelve is 5 st. 6½ lbs., and the average height at thirteen is 4 ft. 9 in. The size of the liver and spleen contrasting with the stunted growth makes the abdomen prominent.

In the later stages hæmorrhages may be seen; thus cutaneous petechiæ, epistaxis, and bleeding from the gums are not uncommon. Hæmatemesis is rare. There may be a simple secondary anæmia—that is to say, a diminution of red cells with a proportionate diminution of hæmoglobin. There is no leucocytosis, or, if present, it is slight. The urine contains bile but not albumen; sometimes there is slight polyuria. There may be a brown staining of the skin, like that of Addison's disease, and this may be an early symptom appearing even before the jaundice. In rare cases a slight enlargement of the lymphatic glands accessible during life may be detected, and more commonly slight enlargement of the lymphatic glands in the portal fissure and neighbourhood of the liver is found after death.

The outlook is grave, for as far as we know the disease, although slow, is always fatal. There is no special treatment, but the patient's general health should be maintained at as high a level as possible. Some have drained the gall bladder and apparently a certain amount of improvement has resulted.

We know nothing as to the cause; sometimes more than one member of the same family is affected, suggesting a common cause for each affected. The disease is not hereditary nor is it due to alcoholic drinks. Those who believe that the cirrhosis starts from the fibrous tissues around the smaller bile ducts suggest that an inflammatory process spreads up the bile ducts from the duodenum; but at a post-mortem the duodenum and bile ducts are usually healthy and so is the pancreas, which we should expect to find diseased if a chronic inflammation spreads from the duodenum.

The diagnosis of this form of cirrhosis is not difficult; the chief points which will lead us aright are the early appearance of jaundice, the size of the spleen, the age of the patient, the absence of a history of over-indulgence in alcohol, and in many cases the stunted growth and clubbing of the fingers. The points of distinction from Banti's disease will be mentioned directly.

Splenic Anæmia.—This is a condition for much knowledge of which we are indebted to Osler. It is sometimes called Banti's disease, after the

Italian physician Banti, who first described the terminal stages of it. Osler defines the disease thus: "A chronic affection, probably an intoxication of unknown origin, characterised by a progressive enlargement of the spleen, which cannot be correlated to any known cause, as malaria, leukæmia, cirrhosis of liver, &c. (primary spleno-megaly), anæmia of a secondary or chlorotic type, leukopænia, a marked tendency to hæmorrhage, particularly from the stomach; and in many cases a terminal stage with cirrhosis of the liver, jaundice, and ascites (Banti's disease)." It is the fact of the terminal cirrhosis which justifies a short description of this disease here. The huge size of the spleen with subsequent cirrhosis of the liver has led to this disease being termed "spleno-megalic cirrhosis," and this name should be used for no other condition; but it will be noticed that as it is only in the terminal stages that the patient has cirrhosis of his liver, he can hardly throughout the whole of his illness be said to be suffering from spleno-megalic cirrhosis. It will also be noticed that there is no Banti's disease apart from splenic anæmia. Banti only described the terminal stages. So we have a disease, splenic anæmia, which in its later stages has, owing to the supervention of cirrhosis of the liver, had the terms spleno-megalic cirrhosis and Banti's disease applied to it.

Splenic anæmia is a disease of which we do not

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know the cause. It is commoner in males than females; some of the patients are children, and usually the sufferers from it are under forty, often about twenty years old. The last patient under my care was a boy aged eleven years. A few instances are recorded of several cases in one family; a brother and sister, both suffering from it, died in Guy's Hospital. It is very long-lasting; cases are known in which it has been present over twenty years. Among Osler's series of fifteen cases, in seven the duration of the disease was more than ten years, and in connection with this it must be remembered that often when the patients first come under notice the spleen is already much enlarged, and therefore probably the disease has then existed some time. The spleen ultimately becomes enormous, larger perhaps than in any disease except spleno-medullary leukæmia. In the case of the boy aged eleven, which I have just mentioned, it weighed 2 lbs. 9 oz. Being so large, it is easily detected during life and the notch is readily felt. A remarkable feature of the disease is hæmatemesis. Among nineteen cases collected by the American Association of Physicians, seven vomited blood, and authors seem to agree that hæmatemesis may occur before cirrhosis of the liver sets in, for when the abdomen has been opened to remove the spleen in cases in which hæmatemesis has occurred the liver has been seen to be healthy. There are probably no diseases, except gastric ulcer

and gastrostaxis, in which hæmatemesis lasts for so many years as it may in splenic anæmia. The patients may also bleed from the nose and gums into the skin, and I have seen hæmaturia.

The blood in splenic anæmia shows the patient to be suffering from a secondary anæmia with sometimes a leaning to the chlorotic type. Usually the red cells are diminished to between 3,000,000 and 4,000,000, but they may fall to 2,000,000 or may not reach 4,000,000. The hæmoglobin is commonly diminished rather more than the red cells, so that a colour index of 0·8 or 0·9 is often seen. In many cases there is a considerable lessening of the number of white cells. Beyond this there is no change in the blood. A few of the patients have had some brown pigmentation of the skin.

Not many cases have been followed from beginning to end, but there appears no doubt that, in the last stage, cirrhosis of the liver supervenes; the case then becomes one of spleno-megallic cirrhosis or Banti's disease, and hence the justification for describing it here. In a very few instances ascites, and less often jaundice, have been associated with the cirrhosis.

At the post-mortem examination on the boy aged eleven the liver weighed only 26 oz.; it looked exactly like a hobnailed atrophic cirrhotic liver. There was much cirrhosis, chiefly intralobular, but considerably multilobular; there was very little intra-

cellular fibrous tissue. During life the liver in this case had been felt an inch and a half below the ribs, and generally when cirrhosis supervenes the liver can be felt hard and, later on, hobnailed. There is great hyperplasia of the splenic pulp with increase of fibrous tissue. In addition there is considerable endothelial proliferation in the splenic blood sinuses.

No drug is known to do any good. Several cases have been recorded in which benefit has been said to follow excision of the spleen. This is done because it is thought that the disease of the spleen leads to a general toxæmia; the cirrhosis, in particular, has been ascribed to absorption of toxins from the spleen. Armstrong collected thirty-two cases in which the spleen was removed; the mortality was twenty-eight per cent. But we must remember that the disease may last for years, even twenty, and it may be slow even after cirrhosis has supervened; I have seen cases in which the liver was felt to be enlarged two years before death. Therefore, as many of the cases of removal of the spleen are comparatively recent, it is clear that we do not know if life is prolonged by this operation, and, even if it is, we must remember that the operation has a high mortality. It is fair to add that individual patients are reported to have been benefited by the removal.

The spleen is enlarged in so many diseases that

the diagnosis of splenic anæmia before the super-vention of spleno-megallic cirrhosis is very difficult, but the difficulties are more properly discussed in works dealing with diseases of the spleen. When the stage of hepatic cirrhosis is reached, the difficulties are even greater, and the opinion that the case is one of splenic anæmia which has passed into the stage of spleno-megallic cirrhosis, or Banti's disease, cannot be expressed with certainty unless we know from the history of the patient that he has suffered from splenic anæmia for some time, because in both ordinary cirrhosis and Banti's disease the liver is cirrhotic, the spleen is large, hæmatemesis may be present, the patient may be jaundiced and suffer from ascites, and not all patients with ordinary cirrhosis have taken alcohol to excess. Points, however, that should weigh with us are that the spleen in spleno-megallic cirrhosis is usually more enlarged than in ordinary cirrhosis; jaundice and ascites are common in ordinary cirrhosis, but probably rare in spleno-megallic cirrhosis; hæmatemesis, when present in spleno-megallic cirrhosis, is more spread over a long period than in ordinary cirrhosis. Still the diagnosis of spleno-megallic cirrhosis should only be made after exercising the greatest care. I have frequently seen the diagnosis wrong, and for a certain diagnosis a correct history is essential.

Obstructive Biliary Cirrhosis. — Some experimenters claim to have produced cirrhosis, spreading

from the bile ducts, by ligature of the common bile duct, but others state that it does not follow. It is very doubtful whether cirrhosis following obstruction to the duct is of any interest in clinical medicine, for it is quite certain that such cirrhosis is in the human subject excessively rare, although obstruction to the duct by gall-stones, malignant disease of the pancreas, and glands in the portal fissure enlarged by malignant disease is very common. Indeed, it is so rare to see cirrhosis in the liver when the bile duct is obstructed that when it exists it is probably not due to the obstruction. I have only twice seen cirrhosis associated with obstruction. Ford, searching through the Surgeon - General's Library at Washington from 1882 to 1900, could only find in those years twenty-one cases of cirrhosis of the liver associated with obstruction to the bile duct, and he adds three cases he himself observed. Of these twenty-four cases in nine there was congenital obliteration of the bile duct, so whatever may have been the cause of the cirrhosis, it is of little clinical interest, for the infants cannot have lived long, and in one there was congenital cystic disease of the liver. He admits that in some of his other cases the cirrhosis may be ascribed to excessive drinking. We need not, therefore, further discuss obstructive biliary cirrhosis, for there is no evidence that it plays any part in clinical medicine.

Cirrhosis in Young Children in India.—Gibbons and Ghose state that a variety of cirrhosis, which is at first pericellular, and later monolobular and multilobular, exists among the young native children of India. It begins at about the seventh month and is usually fatal. The spleen is enlarged and jaundice intense.

Malarial Cirrhosis.—One French author enumerates nineteen varieties of malarial cirrhosis, but there is no such disease. The mistake of ascribing cirrhosis to malaria has arisen from the fact that many people with malaria have taken more alcoholic drink than they should; the doctor has kindly wanted to whitewash them, and so has attributed to their malaria, cirrhosis which should be laid to the door of alcohol.

Bronzed Diabetes.—In this condition, which is very seldom seen, the liver is cirrhotic. The patients are usually males past middle life. The liver is enlarged and hard. It feels like a large cirrhotic liver. Dyspeptic symptoms are very common. The patient wastes and feels very languid. Later he becomes pigmented; the colouring of the skin closely resembles that due to arsenic. There is no jaundice. The mucous membranes are not coloured. Sugar appears in the urine some time after the pigmentation of the skin; the amount is never great, and it often disappears as the patient nears his end. Ascites and cedema of the feet are in some cases late symptoms,

and the patient usually dies comatose. After death the liver is found to be enlarged and hard, its surface is uneven; there is much increase of fibrous tissue, especially in connection with the portal canals; the organ is of a rusty colour, from the deposition of a pigment which contains iron, and is probably derived from hæmoglobin set free by an excessive destruction of red cells. This colouring matter is most abundant at the periphery of the lobules of the liver, but it may be found in almost all the organs of the body, especially the spleen, abdominal lymph glands, and pancreas. This organ is atrophied, and this atrophy is no doubt the cause of the glycosuria. We know nothing as to the cause of the disease or about its pathology, except that there is a considerable destruction of red cells. It is excessively uncommon. I have never seen it in the post-mortem room, and only once have I seen a patient in whom the diagnosis appeared probable, and in him the sugar was discovered in the urine some months before the pigmentation appeared, so that if he was suffering from bronzed diabetes its clinical course was unusual.

Other alleged Causes of Cirrhosis.—It has been stated that rickets, diabetes, gout, cancer, tubercle, passive congestion, lead, dyspepsia, and scarlet fever will all give rise to cirrhosis of the liver, but this is a misuse of histological knowledge as applied to clinical medicine. Occasionally, perhaps, a slight

increase of fibrous tissue may be detected by the microscope in patients dead of these diseases, but this is never enough to cause the liver to look or feel like a cirrhotic liver, nor are symptoms of cirrhosis ever present. Among these conditions increased fibrosis of the liver is most often seen after passive congestion from disease of the heart or lungs (see nutmeg liver, p. 60); but even then the liver never looks like a cirrhotic liver, nor are there any symptoms of cirrhosis.

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SYPHILIS OF THE LIVER

ACQUIRED SYPHILIS

Secondary Effects.—Many authors refer to jaundice as occurring during the secondary stage of syphilis when the rash is present on the skin. It is, however, so rare that it has been suggested that it is simply ordinary catarrhal jaundice occurring in a patient with symptoms of secondary syphilis. The sufficient objection to this view is that it takes much longer to pass away than the jaundice commonly called catarrhal, and does not diminish unless anti-syphilitic treatment is adopted. We do not know how it is produced; some say that it is due to a syphilitic affection of the mucous lining of the bile duct, similar to that of the skin; others think it is caused by swelling of the portal lymph glands, in favour of which it has been said that it is particularly liable to occur in those in whom secondary affection of the lymph glands accessible to examination is evident. Rolleston takes the view that it is a catarrhal condition of the small intra-hepatic bile ducts, which is merely part of a general syphilitic hepatitis. If there is any

change in the liver it is probably a pericellular infiltration with small round cells like that seen in hereditary syphilis, and when this change is excessive it is stated that it may, in excessively exceptional cases, run on into acute yellow atrophy. We know so little about jaundice occurring in the secondary stage of syphilis, not only because of its rarity, but because patients do not often die during the secondary stage. The treatment of this form of jaundice is by mercury, administered in any of the ways usual for syphilis. Under this treatment the jaundice almost invariably soon passes away.

Tertiary Effects.—Syphilis may in its tertiary stage cause three alterations in the liver: (*a*) an increase of fibrous tissue, often very great; (*b*) gummata; (*c*) lardaceous disease, which will be described elsewhere (see p. 245). I find that taking twenty years on from 1885 there were 95 instances of syphilis causing fibrous scarring or gummata of the liver found in the post-mortem room at Guy's Hospital; in 23 of the 95 gummata were present; in the remaining 72 there was only scarring of the liver by fibrous tissue. Sometimes scarring and gummata were present in the same liver. As adults preponderate very largely over children in the post-mortem room of a general hospital, and fibrous bands and gummata are very rare in congenital syphilis, we may, for all practical purposes, assume that in all these 95 cases the syphilis was

acquired. These figures do not show the full effect of acquired syphilis on the liver, for no doubt some of the cases of hepatic lardaceous disease were due to it. During these twenty years about 9500 post-mortem examinations were made, which shows that, roughly speaking, 1 per cent. of all those who die in a general hospital have fibrous or gummatous change in their livers as a result of acquired syphilis. Until about seventy years ago gummata and syphilitic fibrous changes in the liver were regarded as cancerous. In 1855 Busk translated Wedl's book on pathology into English, and it contained an account of Dittrich's work showing that acquired syphilis affects the liver. Shortly after this Sir Samuel Wilks showed some specimens of a syphilitic liver at the Pathological Society, and it is to him that we owe our present recognition of the fact that syphilis will affect the liver and other viscera.

Morbid Anatomy.—The new fibrous tissue produces an appearance of the liver which is quite unlike that of an ordinary cirrhosis, for, instead of being uniformly distributed throughout the liver, the fibrous tissue that results from syphilis forms great bands, several of which usually start from a common point and cut the liver up into large areas which contain perfectly healthy liver. It will be remembered that in cirrhosis the areas were only the size of hobnail, and the liver tissue in them was

usually fatty and affected with pericellular cirrhosis, or at any rate interlobular cirrhosis. The outline of the syphilitic liver is much altered; scar-like depressions mark the surface—they may be so numerous and deep as to divide the liver up into a cluster of irregular masses, so that in extreme cases the liver has been said to resemble a bunch of grapes—but, although it is nearly always considerably deformed, such extreme deformity is rare. On section these bands of fibrous tissue are found to be white, shining and tough. The centre from which they radiate is composed of hard fibrous tissue. Occasionally bands may be seen that do not radiate from a centre.

Gummata in the liver are exactly like gummata elsewhere. Their size depends upon their age; for they grow to a certain size, sometimes as large as a Tangerine orange, and then they shrink. A single gumma is uncommon; usually two or three are present in the same liver. They form considerable tumours on the surface of the liver, so that a syphilitic liver has been known to bear some resemblance to one affected with growth; but usually the distinction is quite easy in the post-mortem room, for the firm, yellowish, cheese-like appearance of gumma is characteristic. All gummata have a zone of fibrous tissue around them. As time goes on the gumma becomes absorbed, the fibrous tissue grows, contracts, and sends radiating processes into

the liver; no doubt some of the radiating fibrous bands previously described originally sprang from around a gumma, for at the time of death we occasionally see the remains of a gumma under a depression on the surface of the liver, and from it bands of fibrous tissue radiate. On the other hand, such fibrous tissue may occur without any previous gumma in its centre. It is clear that the ultimate deformity of the liver can be extreme, for not only may one part be deeply scarred by fibrous tissue, but at the same time, in an adjacent part, gummata may form tumours. As the right lobe is larger than the left these changes are best seen in it, but when they are extreme in the small left lobe the contraction of these fibrous bands may almost obliterate it. Rarely a gumma softens and has been mistaken for an abscess; still more rarely it calcifies, and the calcification has been known to spread into the fibrous bands. Although for a short distance around a gumma the fibrous tissue spreads and causes a slight pericellular cirrhosis, it must be remembered that, as just stated, the liver between the bands is healthy; perhaps it sometimes undergoes compensating hypertrophy.

Clinical Features.—Acquired syphilis affects the liver oftener in men than in women, in the proportion of 3 to 1, and is commonest between the ages of twenty-five and fifty. Both these facts are what might be expected. In the great majority of cases

syphilitic disease of the liver gives rise to no symptoms. It is found far more often in the post-mortem room than the frequency with which it is diagnosed would lead us to expect. It is a remarkable fact that a gumma of the liver is almost invariably near the surface, and thus there may be some inflammation of the peritoneum over it, and this may be painful. Although the deposition of gummata tends to make the liver large, the contraction of the fibrous tissue tends to make it small, so that the total alteration of bulk is not great; but if the organ can be felt, it will be noticed to be irregular with considerable depressions and lumps on it. When the contraction of fibrous tissue is extreme, the liver may be so small that it cannot be felt. Occasionally it may be very large and irregular, when lardaceous, fibrous and gummatous changes all occur in the same liver. This, however, is exceptional; as a rule the livers which show fibrous and gummatous change are not lardaceous. Many rare symptoms have been described, but it is important to remember that they are excessively rare. For example, a gummatous gland in the transverse fissure, or the contraction of a band of fibrous tissue pressing on the bile duct or the portal vein, has led to jaundice or ascites; but these events are so exceptional that if a patient has an enlarged liver, and is jaundiced or has ascites, he almost certainly has not got a syphilitic affection of his liver; 'probably he has

either cirrhosis or malignant disease. Rarely syphilitic fever is present, and one or two cases are on record in which the size of the spleen, which was lardaceous, led to a diagnosis of splenic anæmia, although the patient really had a syphilitic liver.

It has been said that hepatic syphilis is probably not so often considered at the bedside as it should be, but I doubt this, for we have seen that it is only found in the post-mortem room in 1 per cent. of all deaths at a large hospital, and also that few of the cases show any clinical symptoms; therefore it must be very rare for symptoms observed at the bedside to point to it. I have frequently known the suggestion made that a patient was suffering from syphilis of the liver, and then the diagnosis has been corrected to that of cirrhosis or growth; such correction has always been proved to be correct.

Treatment.—The patient should at first take 5 grains of iodide of potassium three times a day, and the quantity should be rapidly increased until 30 grains three times a day are taken. The risk of iodism may be lessened by prescribing some aromatic spirits of ammonia with the iodide, and by taking it diluted with half a tumbler of water. It is probably wise to give a drachm of liquor hydragryi perchloridi with each dose of the medicine. Pain may be relieved by hot fomentations

or leeches. The treatment of syphilis is very thoroughly carried out at Aix-la-Chapelle, and many patients derive benefit from going there. Those who are persistently forgetful about taking their medicine should, if possible, be sent. If detected during life and properly treated, gummata of the liver disappear. I have more than once known a gumma easily palpable completely disappear in two or three months, and the patients have remained well for the many years they have remained under observation. Indeed, I suspect that the giving of iodide stops the formation of fibrous tissue.

CONGENITAL SYPHILIS

The liver is affected in about half the infants who suffer from congenital syphilis, and the affection is nearly always quite different from that due to acquired syphilis, for it usually consists in a pericellular cirrhosis extending through the whole of the organ. In the earliest stages there is capillary congestion, with small-celled infiltration between the cells; the small cells gradually form fibrous tissue, so that the whole liver becomes traversed with fine fibrous tissue extending between the cells. The liver cells are atrophied and compressed, but do not become fatty. The same changes occur in the spleen. To the naked eye both organs are considerably enlarged. The surface of the liver is

smooth; it feels firm; the colour may be healthy, but it is usually lighter than normal. On section small grey bodies are seen; these are collections of small cells and are due to syphilis, but they look like tubercles. No symptoms are caused by the enlargement of either liver or spleen, and during life we can only diagnose that the liver and spleen are affected by congenital syphilis when we observe the enlargement of these organs in a child affected with other signs of congenital syphilis. It must be remembered that the liver is naturally proportionately larger in children than in adults, but if affected by congenital syphilis it may be very large; it has even reached the iliac crest. In a very few cases jaundice has been observed, and its cause is not certainly known. Ascites is very rare; probably it is due to associated chronic peritonitis.

The outlook is grave; many of the children die; the severity of the illness is directly proportional to the size of the liver and spleen. The treatment is that of the congenital syphilis.

Occasionally the liver is affected by congenital syphilis exactly as it is by acquired syphilis. The sufferers from this form of hepatic disease are usually between ten and twenty years old, and they have often signs of congenital syphilis. Fibrous bands and gummata are formed in the liver, which may be felt during life to be hard, lumpy, and irregular. The last patient afflicted with this disease

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under my care was a girl aged twelve; the depressions and lumps in the liver were easily palpable. A few cases are on record of gummata of the liver in infants, and also a few in which congenital syphilis caused lardaceous disease of the liver and spleen. One such a child, a little more than a year old, was under my care some time ago. The great size of the liver and spleen, together with evidence that the kidney was lardaceous, enabled us to make a correct diagnosis.

PERIHEPATITIS

PERIHEPATITIS or inflammation of the peritoneal covering of the liver is either acute or chronic. The acute form, unless traumatic, is an unimportant part of some other acute process, such as acute peritonitis, hepatic abscess, acute pleurisy, or acute pericarditis, and is therefore of little interest. The patient complains of pain in the region of the liver, especially when he breathes or coughs, and consequently the respiratory movements on the right side are limited. The muscles over the liver are rigid, the organ is tender, and a peritoneal rub synchronous with respiration may sometimes be heard over it. Generally the symptoms are quite masked by the severity of those of the underlying cause, and a diagnosis is never complete unless this cause has been discovered.

Chronic perihepatitis either affects the whole of the peritoneal covering of the liver, or it is scattered in patches on its surface, and then it is called local perihepatitis.

Local Chronic Perihepatitis has many causes; for example it may occur over and around a malignant

or tuberculous growth of the liver ; it may spread from an inflamed gall bladder in a case of gall-stones ; it is found around a gastric ulcer which has become adherent to the liver ; it may occur in association with a gumma of the liver. Local perihepatitis occurring in patches is very common when there is severe backward pressure in pulmonary or cardiac disease. It usually has capsulitis of the spleen associated with it, but this is rarely universal when the perihepatitis is local. The thickenings of the capsule of the liver, which constitute local perihepatitis, are usually firmly attached to the subjacent hepatic tissue. They often have little pits on their surface, and often these are so numerous as to give a meshed appearance to the perihepatitis. Usually there are no symptoms ; occasionally a rub may be heard on respiration, and probably the pain of which some sufferers of heart disease and cirrhosis of the liver complain is due to local perihepatitis.

Universal Chronic Perihepatitis is a very different condition from the local variety ; in it the whole capsule of the liver becomes thick, opaque and white ; hence the name *zuckergussleber* has been applied to the liver by Curschmann, and this is the name by which chronic perihepatitis is known in Germany. This white jacket, which may be a quarter of an inch thick, easily peels off from the subjacent liver, the surface of which is smooth ; and for some unexplained reason it is not uncommon

to find the lower edge of the liver folded up on to the anterior surface of the organ and held there by the thickened hepatic capsule. Fagge mentions a case in which the lower margin of the liver touched a point on the anterior surface that should have been four and a half inches distant from it. As a result of the thickening of its capsule, the lower edge of the liver cannot be felt at all; and if the liver can be made out by tactile examination, the surface at first taken to be the lower edge feels particularly thick and rounded. The upper and lower folds of the posterior ligament become so thickened that they are approximated, and the gall bladder may be completely buried in the thickened hepatic capsule, on the surface of which little pits are often to be seen. If the patient chances to die from some other disease early in the course of his perihepatitis, the liver is covered with a thin layer of white lymph, which easily peels off. The thickened peritoneum consists of fibrous tissue, undergoing hyaline degeneration in parts, and arranged in horizontal laminæ. Doubtless a micro-organism is the cause of the trouble, but it is often impossible to say what particular micro-organism is the offender.

The liver with its thickened capsule generally weighs about the same as a healthy liver; from this we may conclude that the organ is a little atrophied.

Some writers have said that the liver in chronic universal perihepatitis is cirrhotic, but this is cer-

tainly incorrect, and the mistake probably arose from not drawing the important distinction between local and general chronic perihepatitis, for which Fagge's description of the liver is undoubtedly correct. He says the hepatic "tissue is commonly soft, and is very often loaded with fat. It is seldom cirrhotic, but there is sometimes an excess of white fibrous tissue in the course of the large portal vessels." This description certainly agrees with what I have observed myself, and among twenty-two consecutive cases of universal perihepatitis that have occurred at Guy's Hospital, I found the liver was never markedly cirrhotic; its tissue was nearly always soft. All authors are now agreed as to the rarity of genuine cirrhosis in association with universal perihepatitis. When it occurs it is an accidental association, and very few cases are on record. Twice in syphilitic subjects I have known the liver covered by the thickened capsule of universal perihepatitis. As will be seen directly, sometimes chronic renal disease, with subsequent cardiac failure, and sometimes chronic pericardial adhesions are associated with perihepatitis; therefore we may find the liver nutmeg.

The thickened capsule hardly ever exercises pressure on the structures in the transverse fissure. Thus jaundice is scarcely ever seen in a patient with chronic universal perihepatitis, and if it is present it is due to some cause apart from the

perihepatitis, such as gall-stones, alcoholic cirrhosis, or heart disease.

The portal vein is not compressed. I have dissected it carefully in an extreme case of universal perihepatitis without detecting any compression of it; and the ascites so often present with this form of perihepatitis has nothing to do with pressure on the portal vein, but is to be ascribed to the chronic peritonitis, of which chronic universal perihepatitis is a part.

It is fundamental to the proper understanding of this form of perihepatitis to grasp the fact that it is only a part of a general chronic peritonitis, and in this fact we have an explanation of the frequency of ascites and the rarity of jaundice. I took quite indiscriminately from the post-mortem records at Guy's Hospital 40 consecutive cases of perihepatitis; 18 were examples of partial and 22 of universal perihepatitis. Of the 18 cases, 6 were instances of perihepatitis due either to tubercle or cancer, and the thickening of the capsule of the liver appeared to be merely part of general peritonitis caused either by tubercle or cancer; of the remaining 12 only 1 is stated to have had peritonitis, and of the 11 left 8 are distinctly stated not to have had any peritonitis; in the remaining 3 the peritoneum is not mentioned. Turning now to the 22 cases of universal perihepatitis, in only 2 is it stated that there was no peritonitis; in 17 it is

distinctly stated that there was peritonitis, and in the remaining 3 no mention is made of the peritoneum. The peritonitis was always chronic, and was never due to obvious tubercle or growth; it was always of that well-known variety in which the peritoneum becomes thickened and opaque; the omentum is puckered up towards the colon, where it forms a transverse ridge, often mistaken for the lower edge of the liver; the mesentery becomes so shortened that the intestines are dragged back to the spine, and in an extreme case they may be so matted together that they can be removed as one mass, from which it may take hours to dissect them. They may be puckered up parallel to their long axis, so that the distance from the stomach to the cœcum is much lessened. Sometimes the material which mats the intestines together can be stripped off, leaving their smooth serous surface exposed, and thus we see the similarity between this chronic peritonitis and the chronic universal perihepatitis. Ascites is a constant symptom of this variety of chronic peritonitis, and hence as chronic universal perihepatitis is nearly always only a part of this form of chronic peritonitis, we see that ascites must be a very frequent symptom of perihepatitis. This view is strongly supported by the fact that in the only two cases I have come across in which chronic universal perihepatitis occurred without chronic peritonitis there was no ascites. As the general

peritonitis implicates the capsule of the spleen, universal splenic capsulitis is nearly always found associated with universal perihepatitis. It must therefore be carefully remembered that universal perihepatitis is nearly always only part of a chronic peritonitis, not due to tubercle or growth. It is, indeed, the variety which, for want of a better name, is often called simple chronic peritonitis.

In my series of cases of universal perihepatitis, the average age at death was $47\frac{1}{2}$ years; the youngest was 29; the eldest 68. The proportion of males to females was 13 to 8, but others have found the sexes more nearly equal.

The accompanying peritonitis may make it difficult to feel the liver in a case of universal perihepatitis, but if it can be felt the edge is thick, uniform, and projects but a little way under the ribs. Unless the case is one of those very rare instances in which there is no chronic peritonitis, we have all the signs of this condition. Thus masses of thickened peritoneum may be felt, the most usual being an elongated tumour lying transversely across the abdomen above the umbilicus, distinct from the edge of the liver and made of the thickened puckered omentum. The ascitic fluid, which usually collects, gradually makes the abdomen enlarged, and, if the fluid is not loculated, there is bulging in the flanks, the umbilicus protrudes, a thrill can be obtained, and there is a dull note at the sides, which shifts

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when the patient is turned. The puckering of the mesentery draws the intestines back, so that the effused fluid quickly makes the abdomen dull all over. It is very characteristic of this form of peritonitis that the fluid quickly re-accumulates after tapping, and usually it is drawn off several times before the patient dies. I have known three cases in which the abdomen was tapped over thirty times, and altogether nearly 800 pints of fluid were withdrawn in each, in my last case seventy tapplings were performed; but more usually the patient succumbs after a few tapplings, say, between five and ten. The fluid is clear and straw-coloured and contains albumen.

If the patient has granular kidney, or if the pleuræ, pericardium, or mediastinum are affected, the signs of these conditions will be added. A common diagnostic difficulty at the bedside is to distinguish between perihepatitis and cirrhosis with ascites. This has already been discussed (see p. 157).

The treatment of chronic universal perihepatitis resolves itself into that of the accompanying general peritonitis, the prominent feature of which is recurrent ascites. The fluid collects so quickly that repeated paracentesis is necessary, and, if ordinary antiseptic precautions are taken, there is no fear of infection of the peritoneal cavity. To some extent the accumulation of fluid may be kept under by diuretics, and of these I think there is no doubt

copaiba resin is the best. Fifteen grains may be taken three times a day in a cachet. If given in a mixture it must be suspended, but such a mixture is very nasty; indeed, the practical objection to copaiba resin is that it often has to be discontinued on account of the nausea it causes. If this is so, the well-known diuretic pill containing a grain of each of powdered squill, powdered digitalis leaves, and blue pill should be tried.

Associated Conditions outside the Peritoneum.—

In a very considerable proportion, probably well over a half, of the cases of universal chronic perihepatitis the kidneys are granular; nor is it surprising that this should be so, for chronic peritonitis is a recognised complication of interstitial nephritis. As might be expected, considering the cardiac failure that frequently follows interstitial nephritis, some of the patients with this variety of perihepatitis showed evidences of heart disease, and thus it may be that under the thickened hepatic capsule we find the liver to be nutmeg. In four out of twenty-two cases which I collected the patients had gout, and this is of interest in connection with the granular kidney. Six of the patients gave a history of alcoholic excess; what relation this had to the perihepatitis it is hard to say. The chronic peritonitis, of which the universal perihepatitis is a part, is almost certainly caused by micro-organisms, and we know that patients who have taken too much

alcohol offer but a feeble resistance to bacterial infection; but certainly this universal chronic perihepatitis does not result from cirrhosis, for none of these six patients showed any cirrhosis of the liver at death, and this universal perihepatitis is not a recognised sequel of ordinary alcoholic cirrhosis. In three of the twenty-two cases the patients had had well-marked syphilis, and probably this was the cause of the perihepatitis, for in two of the three no general peritonitis was present. So many cases of chronic peritonitis are tubercular, that the suggestion has been made that the tubercle bacillus is the cause of universal chronic perihepatitis; but all the evidence is against this view, for generally the most careful post-mortem examination fails to show tubercular lesions elsewhere or bacilli in the peritoneum.

There is a well-defined group of cases in which chronic universal perihepatitis, with its accompanying chronic peritonitis, is associated with chronic inflammation of the pleura and pericardium. To this group the names polyorromenitis, polyserositis, multiple serositis, multiple progressive hyaloseritis, and Concato's disease have been given. In the more severe examples the connective tissue of the mediastinum is affected as well as the pericardium, so that mediastinitis is present. It is, of course, undoubtedly true that many cases of simultaneous, or nearly simultaneous, inflammation of the chief serous membranes are due to the tubercle

bacillus; others to pyæmic micro-organisms; others are associated with heart disease or renal disease; but when all these have been allowed for there remains a group of which we do not know the cause—although it is probably a micro-organism—and they are the cases to which the above-mentioned names are usually applied. The peritoneum is the serous membrane first affected in most of the cases. There are often considerable adhesions between the liver and diaphragm, and thus the trouble spreads to the right pleural cavity, and it is interesting in this connection to remember that the right pleural cavity is more often implicated than the left. The glands in the anterior mediastinum received lymph from the peritoneal cavity, and thus we have the explanation of the affection of the pericardium and the anterior mediastinum. Kelly has drawn particular attention to another group, in which the pericardium is severely and earliest affected; the two layers of it are then firmly adherent, often there is considerable mediastinitis; these two inflammatory troubles so hamper the flow of blood that the liver becomes nutmeg, and by the time the patient dies we find a considerable degree of nutmeg liver as well as chronic pericarditis and chronic peritonitis. As long-continued venous congestion of the liver often leads to an increase of fibrous tissue in it, the unnecessary and cumbersome name pericarditic pseudo-cirrhosis

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has been applied to these cases. It is clear that in such a case the state of the pericardium may lead to much swelling of the feet.

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MALIGNANT DISEASE OF THE LIVER

SECONDARY CANCER OF THE LIVER

SECONDARY cancer is by far the most common form, not only of malignant disease, but also of tumour of the liver. Thus there were admitted into the medical wards of Guy's Hospital during the years 1888-1906, both inclusive, 244 cases diagnosed at the bedside as cancer of the liver, of which not more than 9 or 10 were primary; 46 cases of syphilis of the liver; 45 of abscess; 43 of hydatid, and 28 of sarcoma. The frequency of secondary carcinoma of the liver is also shown by the fact that at Guy's Hospital, among 9500 consecutive post-mortem examinations, there were 361 examples of secondary deposits in the liver, and of these at least 330 were carcinomatous; in other words, secondary carcinomatous deposits are found in the livers of 3.47 per cent. of all persons who die in Guy's Hospital. Further, our records show that of all persons in whom at death malignant disease is found, 50 per cent. have secondary deposits in their liver, and no organ is more frequently affected with secondary deposits.

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The primary growth is far more often at the periphery of the portal vein than elsewhere. The following table, compiled by Rolleston, shows the situation of the primary growth in 76 cases of hepatic secondary carcinoma:—

| | |
|------------------------------|-------|
| Stomach | 24 |
| Colon | 12 |
| Œsophagus | 10 |
| Pancreas | 8 |
| Gall bladder | 5 |
| Uterus | 4 |
| Breast | 3 |
| Kidneys | 3 |
| Bile ducts | 3 |
| Biliary papilla | 1 |
| Vermiform appendix | 1 |
| Bladder | 1 |
| Ovary | 1 |
| | <hr/> |
| | 76 |

The symptoms of secondary cancer of the liver are due so largely to physical changes in its shape that they will be more easy to understand if we first describe the morbid anatomy of the disease.

Morbid Anatomy.—The patient may die from the primary disease soon after the implication of the liver, and then all that will be seen will be a few nodules of growth scattered throughout the hepatic substance; but in many instances there is an enormous deposit of cancer in the liver by the time the patient dies; indeed, no disease causes so great an increase of its weight. I have known

a cancerous liver to weigh 19 lbs., and heavier livers have been recorded, Christian giving one in which the weight of 33½ lbs. was reached.

The nodules in the liver resemble the growth at the primary seat; at first they are very small, but may grow to an enormous size, so that they vary from being so small as to require a microscope for their detection to being as large as a foetal head. In a well-marked case the liver has nodules of new growth all over it, often best seen on the upper surface, and they are hardly ever to be seen in the substance of the liver unless they are apparent on the surface. The older ones are often umbilicated, and the peritoneum over them is generally a little thickened. They destroy the hepatic tissue, contiguous nodules coalesce, so that in a severe case, as they grow more rapidly than they destroy, the liver is a huge irregular mass of new growth, with here and there a little healthy hepatic tissue looking dark by contrast. The colouring is often very vivid, for the cancerous tissue is yellow from staining by bile or a paler yellow from fatty degeneration, and in parts red from hæmorrhage into its substance. The destruction of liver tissue is so great that it is wonderful that life has continued. The microscope usually shows the remaining liver cells to be healthy except where pressure of the growth has caused a little atrophy, and it may reveal a slight increase of

fibrous tissue between the cells. The demarcation between liver substance and growth is usually sharp.

As just remarked, the hepatic growth resembles that at the primary seat, hence its appearance varies accordingly. It may be hard or soft; but after a time the nodules begin to degenerate in the centre, which therefore becomes fatty and soft, and at the post-mortem may be washed away with a stream of water, leaving a shreddy cavity. If the patient lives long enough, the centre may become a cyst. The softening process may lay open a blood vessel, and then hæmorrhage into the growth takes place; it has been known to burst beyond the growth into the hepatic tissue or into the peritoneal cavity. The softening of the growth, combined with more or less contraction of the fibrous tissue in it, is the cause of the umbilication. The cancerous tissue may block the smaller bile ducts, and this is one cause of the biliary staining, and it may grow into veins and cause ante-mortem clotting in them. The new growth may, by direct continuity, implicate the diaphragm and abdominal walls, to which the liver then becomes fixed; but sometimes prominent nodules on the surface of the liver lead to the formation of cancerous tumours on the peritoneal lining of the abdominal wall, at the spot in contact with the hepatic growth, even when no adhesions have taken place. If the mass of growth is great, it may envelop the gall bladder or pylorus; but

when these structures are involved, the explanation usually lies in the fact that they are the primary seats of the growth, which has extended by direct continuity into the liver. The cancer may spread directly along the round ligament, and thus a nodule may be felt in the umbilicus, and the feeling of this may be of great help in coming to a correct diagnosis.

The lymph glands in the portal fissure are very frequently affected, hence they become hard and large, and usually the cause of the jaundice that so frequently accompanies hepatic cancer is due to their pressure on the bile duct. The lymph glands above the left clavicle may be enlarged, and this, too, may be a considerable aid to diagnosis. If the cystic duct is compressed so that no bile can reach the gall bladder, this is found to be empty, but far more often the common duct is compressed by the growth, and then the gall bladder is much dilated from over distension by bile (see p. 26). It is rare for the growth to spread to the kidneys, supra-renals, pancreas, or bowel. The frequent affection of the liver when the growth is at the periphery of the portal vein is probably to be explained by the fact that the primary growth in the course of its ulceration lays open minute branches of the portal vein; this is of interest as malignant growth is usually disseminated by lymphatics. The description of the wasted appearance

of the body, of the primary growth elsewhere, and of the secondary deposits elsewhere, would be out of place here.

Symptoms.—In more than half the cases the deposit of cancerous nodules in the liver produces no symptoms by which they can be recognised during life, and then, as far as we know, they do no harm. If they cause symptoms, these will exist side by side with those of the primary growth, and thus the symptoms of hepatic carcinoma are frequently associated with those of cancer of the stomach or bowel; but in half the cases in which the liver is obviously affected, the seat of the primary growth cannot be detected during life; then after death it is often found in the pancreas, and usually in its head.

The great frequency of cancer of the pelvic organs and breasts of women explains the fact that the proportion of males to females who die of cancer of the liver is as 3 to 4. The ages of 75 per cent. of all patients with cancer of the liver are between forty and seventy years, rather under 20 per cent. are under forty, and rather over 5 per cent. are over seventy; hepatic cancer is all but unknown under twenty.

Both by percussion and tactile examination the liver can usually be made out to be enlarged. The increase of the liver may rarely be so great that the patient gains a little weight in spite of his general

wasting. The organ may reach far below the umbilicus, and the hepatic dulness may be increased upwards a little; but as the organ naturally grows in the direction of least resistance, the heavier it is the more it tends to fall, so increase upwards of the hepatic dulness is not common. The edge of the enlarged organ can nearly always be felt to move up and down with respiration, but occasionally it is fixed by adhesions. It is quite common, when the patient becomes much wasted, for the movement of the enlarged liver and the outward bulging of the lower ribs to be easily visible. It was so in the last patient under my care, in whom a huge tumour, reaching nearly to the iliac crest, could be seen to go up and down with every breath. The edge of the liver feels hard, and, owing to the presence of carcinomatous nodules, is often irregular; the nodules can be felt in so much of the anterior and upper surface as comes below the ribs, so that the whole organ feels irregular, knobby, and hard, and sometimes the lumps on it can be felt to be umbilicated; this is absolutely diagnostic of cancer. Occasionally, if much softening has occurred in them, a faint sensation of fluctuation may be detected, and in a few instances a little local peritonitis causes a rub. If cancer of the liver is suspected, and the lumps on the liver are not at first tangible, the patient should be made to take a deep breath, for there is the possibility that a hepatic tumour usu-

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ally under the ribs may then come down low enough to be felt. Sometimes the cancer grows so fast that an increase of size in the liver is quite noticeable in a week, and rarely a cancerous tumour in the liver may enlarge suddenly from hæmorrhage into it. If either of these rapid alterations takes place the diagnosis of cancer is certain, but in a few instances the nodules get smaller as they undergo degeneration. It must not be forgotten that not all livers enlarged from malignant disease have palpable nodules, for they may be in such a situation that they cannot be felt, or they may be too small to be felt, or the growth may be diffused through the whole liver.

Probably rather more than half the patients suffer from pain in the region of the liver. It varies much in intensity, and some instances are due to local peritonitis. It may be referred to the right shoulder and may go down the right arm. If the liver is very large the patient experiences a sense of fulness and dragging in the right hypochondrium.

About half the patients who during life show symptoms of carcinoma of the liver are jaundiced. In most cases this is due to enlargement of the glands in the portal fissure, but sometimes enough of the hepatic ducts in the liver are compressed, and sometimes when the primary growth is in the head of the pancreas the jaundice may be due to this. It is extremely important to remember that by far the most frequent cause of long-standing

jaundice is cancer of the liver, which produces deeper jaundice than any other common disease; thus patients with hepatic carcinoma present in the most extreme degree those symptoms due to circulation of bile in the blood and its absence from the intestines (see p. 33). The jaundice, too, is permanent, although it may vary a little from day to day. The skin, deeply and slowly stained by bile, gradually becomes a darker green, and is finally of an earthy dark-green tint, which, if the patient be aged and wasted, is almost diagnostic of cancer. The urine is very dark and has a yellowish froth; if there is much itching this is revealed by the numerous scratch marks, the tongue is dry and furred, and a bitter taste in the mouth is often very troublesome, the sweat may very rarely be stained yellow, and if the patient gets bronchitis or pneumonia the expectoration is yellow. Sometimes the pulse is slow, and in very rare instances patches of xanthelasma appear, and still more seldom the patient suffers from xanthops or yellow vision. The usual cause of death is poisoning by bile or, as it is commonly termed, cholæmia. When this sets in the patient becomes more and more drowsy with, in rare cases, an occasional convulsion; day by day his coma slowly deepens, his breathing becomes more and more shallow; at last he cannot be roused, and sometimes for days before he passes away a superficial observer might think that death had already

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taken place. There are few things more characteristic in medicine than to see an aged grey-haired patient extremely wasted, with his dry, dark-green skin hanging in loose folds, lying perfectly still, so drowsy that he is more dead than alive. If we turn down the bedclothes the liver may be seen deforming the shape of the abdomen, and it will be noticed that the sheets are stained yellow, commonly by urine or very rarely by sweat. The absence of bile from the intestines causes indigestion, the motions are pale, they smell horribly, and contain much undigested fat.

As already mentioned under the heading of morbid anatomy, in a few cases the umbilicus may be affected with growth, and the glands above the left clavicle may be enlarged by secondary deposits in them. Pressure by the enlarged portal glands may cause the gall bladder to be distended, but usually the enlargement of the liver prevents its being felt.

Ascites is probably a little less frequent than jaundice. Even when present, the patient often dies before the quantity is large, so paracentesis is hardly ever necessary. Jaundice and ascites are associated in about 20 per cent. of the cases diagnosed during life. The ascitic fluid is usually clear, if jaundice is present it is stained yellow, and if the growth has bled into the peritoneum it contains blood. In the discussion upon the cause of

ascites in cirrhosis of the liver, I have shown (p. 149) that it is unlikely that pressure on the portal vein is the cause of the ascites, and the same reasoning renders it unlikely that pressure of growth on the portal vein is the cause of ascites in cancer of the liver. Probably the cause of the ascites is the implication of the peritoneum by the growth. As the quantity of fluid increases the pain often lessens, and it may be necessary to dip to feel the liver.

Occasionally the growth extends to the right pleura and sets up right-sided pleuritic effusion; the pleural fluid may be blood-stained. The weight of the liver may hamper the circulation through the vena cava; if so, the dilated superficial abdominal veins will show up as dark blue cords on the wasted green skin. The urine may be loaded with lithates and contain much urobilin; rarely there is indicanuria. Rapidly-growing malignant disease of the liver is often associated with an evening rise of temperature to between 99° and 101° , but I have known it rise to over 102° every evening for weeks. As with carcinoma anywhere, there may be a considerable diminution of red cells and hæmoglobin, together with a slight or moderate leucocytosis and some eosinophilia. Rigors rarely occur apart from complications. As in any wasting disease, thrombosis of the veins of the leg may occur.

Prognosis.—Cancer of the liver is usually fatal

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in less than six months from the time the diagnosis is first made. The disease is sometimes very rapid; the patient whose liver at death weighed 19 lbs., was dead in nine weeks after he first complained, and actually worked till within three weeks of his death. In quite exceptional cases life may be prolonged a year or a little longer; in these slow cases the disease may be stationary for a time, and it is not infrequent for patients to improve a little, and even put on a pound or so of weight when they first rest in bed and are carefully dieted. This, however, should not lead us to give up the diagnosis of malignant disease of the liver, if the general evidence for it is strong, for, as a rule, when the diagnosis has been made after due care it is correct.

Diagnosis.—We must be careful not to confuse tumours altogether outside the liver for an enlarged liver. The mistake is most likely to arise with tumours that lie transversely across the abdomen, and they are: Malignant disease of the stomach, malignant disease of the transverse colon, faeces in the transverse colon, and a great omentum puckered from some form of chronic peritonitis. The lower edge of any of these may be mistaken for the lower edge of the liver, especially as they may move up and down with respiration; usually, however, careful palpation enables us to feel the edge of the liver above the tumour, and there may be a band of resonance between it and the liver. I

have known a renal tumour thought to be hepatic, for a tumour of the kidney may, from its attachment to the liver, move up and down with respiration. The liver may appear large when in reality it is not, for it may have dropped as a part of a general enteroptosis, or it may be pushed down by tight-lacing. It is said that it may be pushed down by a pleural effusion, but this has to be very large before it depresses the diaphragm, for it is easier for it to compress the lung and push the heart to the left. The central fibrous portion of the diaphragm nearly always prevents a pericardial effusion from depressing the liver. A sub-diaphragmatic abscess very rarely depresses it, because adhesions form between the liver and the abdominal wall. A growth of the liver hardly ever causes enlargement of the organ upwards sufficient to be detected during life, so that confusion is not likely to arise between a growth of the liver and the very rare cases in which a large tumour, or a large amount of fluid in the abdomen, pushes the liver up.

Often the diagnosis of cancer of the liver is easy, but if the primary growth cannot be found it may be very difficult, especially if the liver is not enlarged. At the bedside the question nearly always takes this form: Is this patient, who has no decided evidence of any primary malignant disease, and who has an enlarged liver, suffering from malignant disease of it? The liver may be

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enlarged from many causes, but the cirrhotic liver, the syphilitic liver, that enlarged from obstructions of the common duct, have to be especially borne in mind when we are considering whether a case can be one of malignant disease of the liver.

The large cirrhotic liver is uniformly large, and the palpable nodules on the surface of it are small. Sir William Jenner used to teach that if any of them feel bigger than a cherry the case cannot be one of cirrhosis; hobnails are never umbilicated and they do not increase suddenly in size, as a malignant nodule may do, from hæmorrhage into it, and the liver never increases in size so rapidly in cirrhosis as it may in cancer; but, on the other hand, it does not in cancer shrink as it may in cirrhosis. Although jaundice is often absent in both malignant disease and cirrhosis, yet when present it may lead us to a correct diagnosis, for when a patient with a large cirrhotic liver has jaundice, it is not commonly very deep, and it always remains yellow; but in cancer it soon becomes intense, and slowly changes to the characteristic deep dirty-green colour already described. Speaking generally, the patient with cirrhosis is more likely to die rapidly after the onset of ascites than is the sufferer from cancer. The obstruction to the outflow of bile is never enough in cases of cirrhosis to lead to a clay colour of the motions, or to dilatation of the gall bladder or bile ducts. The spleen is frequently enlarged in

association with cirrhosis, but rarely with cancer. Extreme wasting and dryness of skin may be present in cirrhosis, but are more frequently met with in cancer. A moderate leucocytosis is often present in cancer, but not in cirrhosis. Although both cirrhosis and cancer of the liver may cause pyrexia, a considerable degree is in favour of cancer. The discovery of either an enlarged gland above the clavicle or of the seat of the primary growth is, of course, proof that the patient has cancer. The age of the patient and an account of his habits will, of course, be a great help.

Syphilis of the liver is rare in the post-mortem room and is still rarer clinically (see p. 191). Still difficulties of diagnosis may arise. The syphilitic liver never attains the great size that may be reached by a cancerous liver, unless it is affected with lardaceous disease, and a lardaceous liver only resembles a cancerous liver when, in addition to the lardaceous change, gummata are present also; this, however, is a very unusual association. Although, as shown in the article on syphilis of the liver, jaundice and ascites may be present, this is so excessively rare that the presence of either is almost proof that the case is one of malignant disease, as is also enlargement of the gall bladder. Other points of distinction are that we never find in syphilis the rapid enlargement of the whole liver or its nodules that may occur in cancer; on the other

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hand, in cancer we never get the marked diminution of both that we may find in syphilis when iodide of potassium is given. Pain and tenderness are much more striking in cancer than syphilis. Rapidity and great severity of the general symptoms are much in favour of cancer. Enlargement of the glands above the left clavicle points to malignant disease, and pyrexia and a slight leucocytosis are more often seen in cancer than in syphilis.

c/ Cases in which, owing to non-malignant obstruction of the duct, bile is retained in the liver, causing it to be enlarged and the patient to be jaundiced, sometimes give rise to great difficulty. Such retention is mostly due either to an impacted gall-stone or inflammatory thickening about the common duct, usually caused by gall-stones. The dilatation of the hepatic ducts may be enormous, and the pancreatic duct may also be greatly enlarged. But these patients rarely have the extremely wasted look, with dry, shrivelled skin, that is so frequently seen with cancer; the hepatic enlargement is uniform, and never so great as it often is in cancer; the jaundice does not become dark green. If it disappears for a time it means that the gall-stone has shifted; that the jaundice of cancer should disappear is almost unknown. Severe obstruction to the duct from inflammatory adhesions is very rare, and a gall-stone is nearly always ultimately passed, for it is most

commonly impacted in the wall of the duodenum and ulcerates into this. The age, the history, and the detection of malignant growth elsewhere will be of help. As far as my experience goes, when we are in considerable doubt as to whether a patient c/ has an impacted gall-stone or a malignant growth, exploration, if done, almost always reveals a growth.

Hydatid tumours of the liver are seldom confused with cancer of it, for almost always they are only one or two in number; the liver is smooth and regular, it is not tender; the hydatid tumour causes neither pain, jaundice, ascites, nor general emaciation, and it may give a thrill. There is no ordinary leucocytosis, but the patient often has eosinophilia. Jaundice is excessively rare, for it would only be caused by rupture of the cyst into the duct, or by pressure of the cyst on it. The exogenous form of hydatid may form multiple tumours, and these and the multiple tumours formed by the alveolar variety would, if they happened to press on the common duct, cause jaundice, but such an event is almost unknown.

Treatment.—At present we know no means by which to cure cancer of the liver. It may be necessary to give morphia for the pain. The treatment of the pruritus is described on p. 34. Constipation and vomiting should be treated on ordinary principles. Washing out the stomach often relieves the vomiting. Ascites rarely requires paracentesis. The

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attempt has been made in a very few cases to excise a cancerous tumour of the liver, but it is virtually never permissible to do so.

PRIMARY CANCER OF THE LIVER

In former times primary carcinoma of the liver was thought to be much commoner than it really is. Thus Frerichs gives the proportion of primary malignant disease of the liver to other cases of malignant disease of the liver as 1 to 5. As will be shown in a minute, this is much too high, and the mistake probably arose from regarding instances of primary malignant disease of the gall bladder and bile ducts as primary cancer of the liver. Primary malignant disease of the bile passages is by no means uncommon. N. F. Ticehurst found that among 11,031 post-mortem examinations at Guy's Hospital there were 45 examples of primary carcinoma of the gall bladder. In 43 out of the 45 cases, that is to say, in 95 per cent., gall-stones were present. These figures strongly suggest that gall-stones are the cause of the cancer, a view which is supported by the fact that the incidence of the age and sex in cases of gall-stones and cancer of the gall bladder is the same; sometimes a definite history of gall-stone colic precedes that of cancer, and often an examination of the gall bladder post-mortem indicates that the gall-stone preceded the

cancer. Analysing the post-mortem statistics at Guy's leads to the conclusion that in about 20 per cent. of the cases of gall-stones, carcinoma of the biliary passages will follow, and this suggests that gall-stones should be much more often removed than is commonly done; and, inasmuch as patients who are jaundiced bear operations badly, it appears that the right thing to do is to remove them at a time when they are not causing biliary obstruction. The danger of gall-stones as a cause of cancer is not confined to the gall bladder, for Ticehurst found among the same 11,031 post-mortem examinations 15 cases of primary carcinoma of the bile ducts, and in 75 per cent. of these gall-stones were present. In former times probably all these cases of primary cancer of the gall bladder and passages would have been regarded as examples of primary malignant disease of the liver. No case should ever be regarded as an instance of primary malignant disease of the liver until a most thorough post-mortem examination has shown that gall bladder and biliary passages are not affected, and that there is no primary seat in some distant part of the body which during life gave no signs to indicate its presence.

I find that out of 18,500 post-mortem examinations at Guy's Hospital, there were 24 undoubted cases of primary malignant disease of the liver. In order to be on the safe side I have rejected all that

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had any growth anywhere than in the liver, unless it was a small nodule or two in the adjacent lymph glands, or a small nodule on the base of the right lung, for these are the most likely seats at which deposits may occur secondary to primary malignant disease of the liver. Twenty-four cases among 18,500 post-mortem examinations show that 0·13 per cent. of all persons who die in a large hospital succumb to primary carcinoma of the liver. The ratio of undoubted primary to secondary carcinoma of the liver is about 1 to 21.

There are three forms of primary carcinoma of the liver. The most frequent is that in which the new growth is deposited in nodules, and the liver exactly resembles the organ which is the seat of secondary deposits. About 65 per cent. of the cases fall into this group. The second form is often called the massive variety of primary hepatic carcinoma; in it the cancer forms one large mass in the liver. Bright describes a case, saying that "the tumour within the liver was the size of an adult's head and of rounded form." In a few instances of this variety there have been one or two much smaller similar tumours in other parts of the liver. About 23 per cent. are of this variety. In the third form, constituting about 12 per cent. of all primary malignant hepatic tumours, the cancer cells are uniformly diffused through the liver, and there is great increase of fibrous tissue in all direc-

tions. This may contract, and then the liver, although at first larger, ultimately becomes smaller than natural. In three cases we had at Guy's the weights of the livers of this third variety of primary hepatic carcinoma were 120 oz., 62 oz., and 36½ oz. The contraction of fibrous tissue in the last must have led to considerable atrophy of hepatic tissue. The fibrous tissue in these cases is very dense and hard, and runs through the whole of the liver. Hæmorrhage into the cancer is very rare and so are degenerative changes. The glands in the portal fissure are rarely affected, secondary deposits are almost unknown, and the cancer hardly ever grows into the portal vein or bile ducts. Indeed, everything points to the fact that the patient dies soon after the beginning of the disease, which is rapid. It is often extremely difficult, when looking at a liver affected with this form of carcinoma, to say whether it is a cirrhotic or carcinomatous liver; indeed, it may be almost impossible to say for certain even when looking at microscopical sections.

The following account of the leading features of primary carcinoma of the liver is the result of analysing the cases that have occurred at Guy's Hospital, and these collected by Eggel. A few quite exceptional cases have been recorded as occurring in children; but, apart from these, primary carcinoma of the liver is a disease of adult life and usually of old age. Considerably over half the

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patients are males, a point of great interest, for we have seen that secondary hepatic cancer is commoner in women than men. In rather over a third of the cases that have occurred at Guy's Hospital the temperature ranged between 101° and 102° , and others have recorded cases in which a moderate degree of pyrexia was present; and it is worthy of further investigation to see if the temperature is higher and more often raised in primary than in secondary carcinoma of the liver.

Jaundice is present at some time or another in about half the cases, but it usually comes on late and is not nearly so deep as it commonly is in secondary carcinoma. Probably we seldom see in the primary form the deep, dark olive-green jaundice which is so characteristic of secondary hepatic cancer. The explanation of this may be that as the disease is rapidly fatal there is no time for deep jaundice to supervene. There may be jaundice in cases in which the portal glands are normal, then it must be dependent upon obstruction to the bile ducts in the liver by the hepatic growth. About half the patients suffering from this disease have ascites, and in many of them it may be detected during life. Occasionally the growth invades and causes thrombosis of the portal vein. The patient is often anæmic, and, as with other anæmias, swelling of the feet may be present.

Except in the very few cases of the third variety of primary carcinoma in which there has been much contraction of fibrous tissue the liver is enlarged. In one case the enormous weight of 30 lbs. was reached, but anything over 10 lbs. is very rare, and a common weight is between 120 and 130 oz. The liver can be made out to be enlarged both by percussion and palpation; the feel of it will depend upon the variety of carcinoma that is present. As might be expected, the right lobe is more often affected than the left.

Prognosis.—The average duration of the cases at Guy's was twelve weeks, and from reading accounts of these and other cases I think it may be safely said that primary malignant disease of the liver is usually rapidly fatal, thus forming a striking contrast to those cases in which the organ is affected secondarily, for then the patient often lingers for a long time.

It appears, therefore, that primary cancer of the liver resembles the secondary form in many particulars, but it is more rapidly fatal; it is commoner in men than women; pyrexia is perhaps higher and more frequent; jaundice is seldom deep olive-green, and, if present, is usually slight; and the motions are rarely pale.

An interesting case that occurred at Guy's Hospital shows the difficulties of diagnosis which may arise. A woman, aged thirty-nine, was admitted for what

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was thought to be the vomiting of pregnancy. There was no jaundice, and disease of the liver was not suspected. Premature labour was induced, but the patient sank. The post-mortem examination revealed the fact that the liver was the seat of extensive malignant growth, but all the other organs of the body were absolutely normal.

Three cases at least are on record in which a primary carcinoma of the liver originated in an adrenal rest.

Primary malignant disease occasionally takes place in a liver which is already cirrhotic, and sometimes this is said to be the fourth variety of primary hepatic carcinoma. For example, a man aged forty-one was admitted into Guy's Hospital. He had drunk hard and had ascites and right pleural effusion. When the ascitic fluid was drawn off a lump was felt in the liver. He was never jaundiced, and died three days after the paracentesis. The liver weighed 118 oz., and there was extreme cirrhosis in the parts unaffected by the growth, which consisted of a large cancerous mass in the right lobe, together with smaller masses in other parts of the liver. It was a spheroidal carcinoma. Secondary malignant disease may also occur in a cirrhotic liver, for I once made a post-mortem examination on a man who died of sarcoma of many bones. There was a secondary growth in the liver which weighed 60 oz. and was very hard and cirrhotic.

Sarcoma of the Liver is either primary or secondary. The primary form cannot be distinguished clinically or by the naked eye after death from primary carcinoma of the liver. Indeed, the distinction is often difficult even when examining the organ histologically, for I have known different opinions upon the same section given by competent histologists. Primary sarcoma of the liver is of extreme rarity. Many of the cases occur in children, but the disease may be seen at any age. As with primary carcinoma, there are three varieties, one in which numbers of growths are scattered throughout the liver, a second in which the new growth forms one large mass, and a third in which the sarcomatous growth infiltrates the whole liver. A primary sarcoma of the liver which weighed nearly 17 lbs. is recorded in the Pathological Society's Transactions.

Secondary sarcoma of the liver exactly reproduces the original growth. Secondary hepatic sarcoma is rarely suspected during life, for the patient usually dies from the effect of the primary growth before the affection of the liver produces clinical symptoms. The primary seat is most often in the bones, but the mediastinum and supra-renals are common seats. The secondary growths are usually very numerous in various parts of the body, but the liver itself generally contains only one or two nodules.

Pigment Tumours of the Liver.—All pigmented tumours of the liver are either sarcomas or carcinomas,

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and, strictly speaking, do not merit special description. They are always black and are usually called melanotic sarcoma or carcinoma, as the case may be, differing only from ordinary sarcomas and carcinomas in that the cells of the tumours contain abundant black pigment granules. A liver thus affected forms a very striking object, for the black growths show up very effectively against the reddish-brown liver substance. Melanotic tumours of the liver are decidedly rare, are nearly always sarcomatous, and are almost invariably secondary to melanotic sarcoma of either the skin or the eye. Usually the melanotic sarcomata, of which there may be one or dozens, are scattered throughout the liver; very exceptionally the growth is diffuse, forming one mass throughout the liver. A few cases of primary melanotic sarcoma of the liver are on record, and primary melanotic carcinoma has been known to occur. I have seen one case.

Pigmentary malignant disease of the liver has no separate clinical symptoms from ordinary malignant disease; so, unless a primary melanotic tumour is discovered during life, it cannot be foretold that pigment will be found in the hepatic growths, unless, on exposing the urine of such a patient to the air, a brownish or blackish discoloration of it (melanuria) appears.

Adenomata.—These are perfectly well-defined tumours having the same structure as proper hepatic

tissue, except that the cells may be a little larger than usual, often have double nuclei, and may have a slight increase of fibrous tissue between them. The rest of the hepatic tissue is healthy. They are rare and are almost always solitary; there is a specimen in the Guy's Hospital museum showing such a tumour $1\frac{1}{4}$ in. in diameter projecting from the liver of a man, aged twenty-six, who died from a strangulated hernia. A man in St. Thomas's Hospital had a considerable tumour projecting from the left lobe of the liver; it was thought to be a hydatid, but on operation it turned out to be a solitary adenoma. Very rarely in man, but commonly in dogs, adenomata are multiple; they are then quite small.

The term adenoma has been applied to two other forms of solitary hepatic tumours, viz. those derived from a multiplication of bile capillaries and those due to the growth of an adrenal rest; both are exceptional and are chiefly of histological interest. When, in cirrhosis of the liver, the multiplication of liver cells, which is probably compensatory, forms nodules more prominent than ordinary hobnails, they are sometimes called multiple adenomata. Finally, it must be remembered there is often much difference of opinion among experts whether a cirrhotic liver showing these prominent hobnails should be regarded as a case of cirrhosis with multiple adenomata or cirrhosis with carcinoma.

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Lymphadenoma.—New formations consisting of lymphoid tissue, either generally diffused through the liver or occurring in nodules, may be seen in patients dying from Hodgkin's disease or from leukæmia, but they do not occur apart from these diseases.

Angiomata.—It is by no means uncommon to find small cavernous angiomata in the liver in the post-mortem room, but they cannot be detected during life unless they are large enough to give symptoms which result from their size, and this is very unusual. In nearly all the cases in which a large tumour of the liver has been thought to be a carcinoma, and yet the patient has seemed well enough to be suitable for operation, the growth has turned out to be a cavernous angioma and these tumours have been excised by surgeons from the liver. Fifteen such cases are on record, and the patient was usually under fifty years of age.

Minute fibromas are sometimes seen in the liver ; they have no clinical importance. A myxomatous hepatic tumour has been described.

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FATTY LIVER

SOME writers draw a sharp distinction between fatty infiltration of the liver, in which there is an excess of fat in the hepatic cells, and fatty degeneration, in which the protoplasm of the hepatic cells is replaced to a greater or less extent by fat; but inasmuch as the two processes merge one into the other and are often present in the same case, it is wiser to speak of fatty liver, meaning thereby a liver in which there is an excess of fat.

When there is a great excess of fat in the liver it is uniformly enlarged, and may in, for example, phosphorus poisoning, weigh 10 or 12 lbs.; but sometimes, if there has been much atrophy of the liver cells, a liver may be very fatty and of normal, or less than normal, weight. A fatty liver is pale and soft, its surface is uniform, its edges are rounded. On section it is light yellow, and it readily breaks down when the finger is thrust into it; in an extreme case the knife that cuts it becomes greasy, a piece of such a liver may burn with a flame, will float on water, and leave a greasy stain on a piece of paper. Under the microscope

droplets of fat are easily seen ; they are for the most part at the periphery of the hepatic lobule. If fatty degeneration preponderates, the droplets of fat are usually smaller than in fatty infiltration, and they are more scattered throughout the lobule.

There are many causes of fatty liver. Often it is due to some poison, thus it is frequently met with in those who have taken too much alcohol, and cirrhotic livers often contain a considerable excess of fat. Phosphorus, arsenic, antimony, chloroform, iodoform, sulphonal, and many acids cause fatty liver ; so do bacterial toxins, but not to such an extreme degree as some of the poisons just mentioned ; thus it may be associated with typhoid fever, pneumonia, puerperal fever, cholera, streptococcal infections, diphtheria, scarlet fever, and erysipelas. Tubercle causes, in some cases, considerable fatty change in the liver, and it is a striking thing at a post-mortem examination on a person who is extremely wasted, and in whom all the subcutaneous fat has disappeared as a result of tuberculosis, to find the liver very fatty. Severe anæmia, *e.g.* pernicious anæmia, is often accompanied by a fatty liver. General obesity is often associated with a fatty liver.

A considerable degree of fatty liver may be present, and yet it may not be detected during life, for the obesity of the patient may make it difficult to feel the liver ; and even when there is not an

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excess of fat in the abdominal wall, the liver is difficult to detect, as it is soft. When, however, a fatty liver can be made out, it is felt to be uniformly enlarged, to have a rounded edge, and to be soft; the enlargement is painless, the hepatic dulness may be increased. Addison pointed out that the skin of those who have a fatty liver is pale and smooth, looking like polished ivory or satin. This is best seen in the fingers. As far as we know, a fatty liver does not give rise to any symptoms, but those whose liver is fatty bear accidents, operations, and severe illnesses, as pneumonia, badly.

LARDACEOUS DISEASE OF THE LIVER

AMONG those who suffer from long-continued supuration or from syphilis, several organs of the body undergo a peculiar change, commonly called lardaceous or waxy. The spleen, kidneys, and liver are most often affected, and usually more than one organ is affected in the same patient; in a severe case many organs are affected, *e.g.* kidneys, liver, spleen, intestines, large blood vessels, lymphatic glands, bones, supra-renals, and thyroid gland. The central nervous system is certainly, and the muscular system and skin are probably, never affected; the genitals, bladder and lungs hardly ever. Rolleston, adding together the figures from many authors, found that in 795 cases of lardaceous disease the spleen was affected 585 times, the kidneys 539 times, and the liver 387 times.

The chemistry of lardaceous change is not properly understood; there is a peculiar metamorphosis of the proteids of the affected tissues, but local conditions must be of account, for not all the proteid tissues, *e.g.* the muscles, are equally affected. Nor is it known what is the precise cause of

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lardaceous disease in those who suffer from prolonged suppuration and syphilis. The suppuration must be prolonged, and therefore lardaceous disease is not often seen in these days of antiseptic surgery; but in olden days it was quite common, and was the cause of death in many cases of, for example, chronic disease of the hip, which often succumbed to either the tubal nephritis, which prolonged lardaceous disease of the kidneys sooner or later sets up in those organs, or to the diarrhoea which follows lardaceous disease of the intestine. Tubercular disease does not cause lardaceous disease unless suppuration occurs, and even when that happens it is rare. Why it should be associated with some and not with all cases of suppuration in connection with tubercle we do not know, but it is one—although an uncommon one—of the complications of phthisis that may prove fatal. Likewise it is a strange circumstance that it is only an exceptional result of syphilis. Perhaps the improved treatment of syphilis has something to do with this. I have seen lardaceous disease of most of the organs of the body in a young child afflicted with congenital syphilis. Lardaceous degeneration may stop if the long-continued suppuration is cured or the syphilis actively treated. I have occasionally found lardaceous disease in the post-mortem room in those who have not suffered from prolonged suppuration or syphilis, but such cases are very few;

some of these patients have had cancer, others leukaemia, but the relationship of these to lardaceous disease is not known.

If lardaceous disease is considerable it can be recognised by the naked eye, but if a little Lugol's solution (iodine dissolved in water containing a little iodide of potassium) is poured over the cut surface of the affected organ, and washed off, after a few seconds, with a stream of water, the lardaceous material becomes a deep mahogany colour, even if but little of it is present. If sulphuric acid or a solution of chloride of zinc are now poured on the cut surface, the mahogany colour turns blue. In microscopical sections treated with methyl-aniline violet or gentian violet, the lardaceous material is stained rose-pink instead of blue, the colour that normal tissues are stained.

The liver, when affected with lardaceous or amyloid disease, is uniformly enlarged. The increase of size may be considerable; indeed, next to cancer, lardaceous disease causes the largest livers with which we meet. The organ may weigh 14 lbs. The surface is smooth and shiny, and the whole organ is hard, as though paraffin or a solidified fat had permeated it, and this appearance gave rise to the names lardaceous and amyloid. The result of this setting or hardening is that in the post-mortem room the liver keeps its shape remarkably, so that the anatomical depressions on it, *e.g.* that made by

the right kidney, are very evident, and Rolleston's description of a lardaceous liver, as resembling the plaster cast of the liver seen in an anatomical model, is very apt. The edge is sharp and hard. When we cut the liver we find that its consistency is much increased; it can be cut into much thinner slices than can the normal liver, the cut surface is shiny, the specific gravity is increased, and, according to Wilks, may reach 1080.

Microscopically it is found that the lardaceous material is deposited in the walls of the vessels. The change, as can be readily seen in a stained specimen, is best marked in the capillaries of the intermediate zone of the lobules, so that they become enlarged and compress the hepatic cells, which, however, themselves never become affected. Probably the change first begins in the middle coat of the smallest arterioles; ultimately the capillaries and the veins are all affected.

Clinically a lardaceous liver is known by the fact that it causes no pain, is uniformly enlarged, is smooth, and has a sharp edge. If it is due to syphilis and it is associated with fibrous and gummatous changes in the liver, this organ may be irregular, but such an association is very unusual. The diagnosis is helped by finding the considerable uniform enlargement of the spleen, which is characteristic of lardaceous disease, by detecting evidence of lardaceous disease of the kidneys or intestine,

and by observing whether the patient is suffering from prolonged suppuration or syphilis. The liver may diminish in size if the suppuration is cured or the syphilis brought under the influence of active treatment. •

TUBERCULOSIS OF THE LIVER

Although there are several varieties of this, it is far from common. New-born guinea pigs often show tuberculosis of the liver if the mother is tubercular, but in most animals, including man, it is excessively rare for the foetus to be born with tubercle bacilli in it, even when they swarm in the mother. Tuberculosis of the human placenta is hardly ever seen, but it does occur. Searching medical literature for twelve recent years I was only able to find six cases of transmission from the human mother to the foetus of tubercle bacilli. When it does occur the foetal liver is the organ most often affected, as might be expected, seeing that the foetal blood is returned by the umbilical vein to the liver; indeed, in such cases tubercle bacilli have been found in the blood of the umbilical vein. Sometimes in these very rare cases tubercle bacilli are found in the foetal liver without any obvious change in the liver itself, but in other cases the infection of the liver has lasted long enough to cause hepatic miliary tubercles.

When a person dies of generalised tuberculosis, miliary tubercles are often seen in the liver. The

bacilli are conveyed to it by the portal vein if the focus is at its periphery, *e.g.* tuberculous ulceration of the intestine; otherwise they are conveyed by the hepatic artery. The miliary tubercles may be seen under the hepatic peritoneum or in the liver substance. They are often very numerous; they are just like miliary tubercles seen elsewhere, and they do not give rise to any clinical symptoms.

Caseous tubercular disease of the liver occurs, but is excessively uncommon. The tubercle bacilli are usually conveyed to the liver by the portal vein. Sometimes a number of small caseous areas, which have broken down to form cavities, are seen, and when this happens the caseous material may be discharged into the bile ducts, just as caseous material from the lungs is discharged into the bronchi. Sometimes bile passing into these cavities stains them green or yellow, or, if hæmorrhage has taken place, they are red. Before these numerous caseating masses break down they may, unless care is taken, be mistaken for nodules of lymphadenoma, or other forms of growth, such as the fatty multiple adenomata seen in cirrhosis. I think confusion has sometimes occurred between tuberculosis and psorospermiosis. This form of tubercle does not give rise to any clinical symptoms. There is never damage to a sufficient number of bile ducts to cause jaundice.

In other cases the caseous masses are large, and, speaking generally, the larger they are the fewer

they are. These large masses are very rare, but I have made a post-mortem on, at least, one case. If the caseous mass is large it does not usually rupture in the biliary channels, probably because it is so large that it destroys them; hence these large caseous masses are very rarely bile-stained. They are sometimes called solitary tubercle of the liver; but usually more than one is present in the same liver.

There is a specimen in the Guy's Hospital museum in which there is a triangular pale area in the right lobe of the liver. The base of this area is three inches long. It consists of hepatic tissue invaded by a speckled yellow deposit, which is caseous and histologically tuberculous. The whole liver is studded with small tuberculous deposits. This specimen suggests that some at least of the large caseous masses are due to coalescence of smaller ones, and it also forms a link between the cases with many small caseous nodules and those with large ones. There is another specimen in which there are only two tuberculous caseating masses, each a quarter of an inch in diameter. Such a specimen illustrates that some at least of the larger caseous nodules are not due to coalescence of several smaller ones. There is another in which almost the whole of the left lobe of the liver has been converted into a tuberculous abscess. In very rare instances, such as the last, the case presents itself as one of hepatic tumour or abscess. Mayo Robson has successfully

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drained one such. These large caseous masses may be confused with a breaking-down gumma or actinomycosis of the liver. They are so rare that they are hardly ever diagnosed, although they may be suspected if a tumour of the liver is found in a person very ill from tuberculosis, especially if there is tubercular disease at the periphery of the portal vein.

ACTINOMYCOSIS (STREPTOTRICHOSIS) OF THE LIVER

SEVERAL species of the genus streptothrix of vegetable parasites are the cause of disease in man and animals. Formerly the name actinomycosis was employed to designate this genus, and men or animals were said to be suffering from actinomycosis of the lung, liver, brain, jaw, &c., according to the part disease of which was caused by the parasite, and the word is still very largely used. But streptotrichosis is also employed to indicate the disease caused by streptothrix infection.

There are many species of streptothrix which cause disease in man and animals; but it has not yet been made out that the clinical symptoms or appearances after death vary according to the species, and as there is a considerable resemblance between the various species when they are examined under the microscope, clinical physicians do not usually attain greater accuracy than to say that a patient has actinomycosis or streptotrichosis of such and such an organ, without stating the particular species of the parasite that is present. But it is

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highly desirable that further work should be done with the object of discovering how far the symptoms and post-mortem appearances vary according to the species of parasite that is present, for they can readily be differentiated by experts. The only streptothrix which has hitherto been found in both man and the lower animals is the streptothrix bovis communis, which has been found to be the offending parasite in a few of the cases in man.

We know little for certain about the sources from which human beings are usually infected, but it is often suspected that the disease is derived from wheat, for there are many cases in which it has occurred among those who chew straw, and actinomycosis is especially common in the structures around and in the neighbourhood of the mouth; indeed, in 57 per cent. of the cases the disease is in the head or neck, and in 21 per cent. in the gastro-intestinal tract or abdominal viscera. The case of a boy who swallowed an ear of barley is recorded; an actinomycotic abscess formed between the spine and the scapula, and the ear of barley was found in it. Three males are affected to one female, and the disease is commonest between the ages of ten and forty; but adult men are just those whose occupation brings them most in contact with wheat.

The disease can be conveyed from man to animals, *e.g.* rabbits, cattle, by innoculating them with pieces of diseased tissues from man, and the

micro-organism can be cultivated from the diseased human tissues and transferred to animals by innoculating them with the culture; but no organism found outside the body has been cultivated and produced the disease in animals after being injected into them.

We only certainly know of the presence of streptothrix in man by the results it produces. The first effect is the development of granulation tissue around the parasites: if they die, as they often do in some parts of the body, the granulation tissue becomes converted into fibrous tissue. But often, and probably always in the case of the liver, some of the granulation tissue breaks down with the formation of pus, other parts of it necrose, other parts caseate, and in other parts the formation of fibrous tissue shows an attempt at repair. Sooner or later the actinomycotic focus becomes infected with other pyogenetic organisms.

The process spreads by the growth of the parasite. Therefore, by the time the liver is examined after death, we see a large area of it converted into a mass of purulent, caseating, necrotic tissue, with numbers of fibrous trabeculae running through it and making a number of small cavities filled with thick purulent material. Many of these cavities communicate with one another, but so irregularly that complete drainage of the whole is impossible, and the affected part of the liver looks like a sponge

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soaked in pus. Usually there are, at a little distance from the main mass, smaller similar areas in the hepatic substance. The pus nearly always contains yellow bodies about the size of a pin's-head; these are easily seen, and, when present, are diagnostic. They consist of masses of the micro-organism imbedded in a collection of pus cells, and, when magnified a few times, look like minute raspberries. Magnified still further, we see the innumerable interlacing filaments of the mycelial mass, and the spores of the species of streptothrix, which is the cause of the disease in the case examined. Sometimes the ends of the filaments are rounded like an Indian club, but this clubbed arrangement is commoner in cattle than in man; and often the filaments radiate regularly like rays from the central mycelial mass, hence the name "ray fungus." If some of these rays are clubbed at their free ends and other filaments are unclubbed, the whole appearance is symmetrical and striking. Sometimes the filaments are so short that they are all club. Dr. Acland states that out of 47 cases the threads or filaments were present in 34, the clubs in 24, and in 11 both clubs and threads were present. Although these appearances are, of course, best seen in stained specimens, they may often be seen without staining. For details of staining, books on bacteriology should be consulted. It ought to be added that in many cases the micro-organism dies out, for it has a low vitality; so that

it may be quite impossible to successfully grow the micro-organism on artificial media, even when numerous filaments and spores can be seen under the microscope.

Probably the various species of streptothrix manufacture no toxins which are harmful, or produce no general symptoms, so that when pyrexia, loss of flesh, anaemia and anorexia are present, as is often the case, these symptoms are due to secondary infection by other micro-organisms. Generally speaking, the various species of streptothrix grow by direct continuity. Thus actinomycosis of the lungs will invade the pleura, next the chest wall, and finally discharge externally. Actinomycosis of the liver may invade the diaphragm and then the lung, and this tendency to spread is well seen in the tissues of the neck. Dissemination also takes place by the blood vessels and lymphatics; hence it is not very rare to find two or more organs affected in the same person. When the liver only is diseased, the micro-organism is probably conveyed to it from the alimentary canal by the portal vein.

Actinomycosis of the liver is happily extremely rare, and could only certainly be diagnosed by finding the micro-organism in the pus, or by observing that the patient had symptoms of disease of his liver and actinomycosis in some other situation, *e.g.* the face; but, inasmuch as localising

symptoms indicating hepatic disease are seldom present, it follows that hepatic actinomycosis is of little clinical interest. The patient usually wastes, becomes anæmic, has anorexia, and suffers from irregular pyrexia, all these symptoms being due to secondary infection. If there were a local increased dulness, local tenderness, or local swelling, pointing to the infection of the liver, actinomycosis of it would be mistaken for an ordinary hepatic abscess until an opportunity for examining the pus arose.

As far as is known, all cases of actinomycosis of the liver have proved fatal; but judging by the favourable results that have followed treatment of actinomycosis in parts such as the skin, in which it is accessible, the right thing to do if actinomycosis of the liver were discovered would be to open the abscess, break down the trabeculæ forming its sponge-like structure, so as to give free exit to the pus, and to swab out the cavity with some antiseptic which would not give rise to symptoms of general poisoning from absorption. This treatment would do much good, for the micro-organism is easily killed, and there seems no doubt but that the cure is hastened by giving large doses of iodide of potassium.

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HYDATID DISEASE OF THE LIVER

THE *tænia echinococcus* is a common intestinal parasite of the dog. It is a small tape-worm, for even large specimens do not exceed five millimetres in length. It usually has four joints. When the parasite is fully grown the last joint is larger than all the others. At the free end of the first segment, commonly called the head, are two concentric rows of hooks; each row contains between fourteen and twenty. Behind the two rows of hooks are four suckers, all at the same distance from the hooks; behind the suckers the first segment narrows before its attachment to the second segment, which is larger than the first. The second segment is often called the first proglottis, the third segment the second proglottis, and the fourth segment the third proglottis. The second proglottis contains imperfect sexual organs, and the large third proglottis contains fully-developed male and female sexual organs, and, when mature, many ova, often 500. These parasites inhabit the upper half of the small intestine of the dog, and one animal may be the host of many hundreds. They lie buried among the villi, and are attached to the

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mucous membrane by the hooklets or by the suckers. By the time the terminal proglottides are ripe the ova contain embryos, each of which has a chitinous capsule, attached to which are six spines. At this stage of development the proglottides break off, and are discharged in the dog's fæces. Their soft tissues decay, and thus the embryos are set free. They then die, unless they obtain entrance into some animal in which they can further develop, and man is such an animal. Perhaps sometimes they are taken into him as dust attached to his food, but undoubtedly generally they gain entrance owing to his drinking surface-water which has become infected with them. From the alimentary canal of man these embryos make their way to the liver, and it is almost certain they do this by burrowing their way with their spines into the minute terminal branches of the portal vein, by the blood of which they are conveyed to the liver; indeed, they have been found in the blood of the portal vein. Each embryo is about three times the diameter of a red blood-corpuscle.

When the embryo arrives at the liver its spines disappear and it undergoes important changes, for it quickly becomes a cyst, having two walls which enclose a fluid centre. When the embryo reaches the cystic stage it is called a hydatid. The outer wall is opaque, white, and laminated, the laminae separating easily like the coats of an onion, and this

opaque, white, laminated membrane is very characteristic of hydatids. As time goes on this outer wall becomes quite thick. It contains much chitin. The inner wall consists of cells. The fluid is colourless, limpid, it does not coagulate on heat, and by this important fact it can be distinguished from all inflammatory effusions, *e.g.* that of pleurisy. It is usually neutral, contains 98·5 per cent. of water, has a specific gravity of 1010, and half the solids are sodium chloride; hence there is an abundant precipitate with silver nitrate, which is another important distinguishing feature of hydatid fluid. It contains a powerful poison, for when injected into animals it causes diastolic arrest of the heart, with lowering of the blood pressure and temperature. The poisonous character of hydatid fluid explains, as will be mentioned directly, the serious results that may follow puncture of a cyst, and also, no doubt, is the cause of the urticaria of which some sufferers from hydatid disease complain when hydatid fluid escapes into the tissues or serous cavities. Outside the hydatid a capsule of connective tissue derived from the liver is formed; often it is extremely slight.

The inner cellular layer of the cyst is the most interesting. Here and there its cells proliferate, and thus little elevations are formed projecting into the fluid. These increase in size, and in the centre of each a cavity forms; this becomes filled with fluid

like that already described. Thus we have projecting from the inner wall of the original cyst a number of secondary cysts, commonly known as brood capsules. The attachment of each of these to the inner wall of the original cyst is very delicate, and therefore after death, or at operation, the brood capsules are often seen free in the fluid of the original cyst, for the manipulation of a post-mortem examination or an operation will easily rupture their attachments. Soon after the brood capsule has formed local thickenings take place on its outer surface; each thickening grows outwards, so that it soon becomes a process having in its interior a longitudinal prolongation of the central cavity of the brood capsule. At the free end of this process suckers and rings of hooklets appear, and a scolex forms—that is to say, a living structure exactly like the first segment of the *tænia echinococcus*, the inhabitant of the dog. The process usually at some period of its existence becomes invaginated into the interior of the brood capsule, so that a fully-formed brood capsule has, projecting from its circumference towards its centre, a number—often between ten and twenty—of scolices. The number of brood capsules in one original cyst may be many thousands. After a brood capsule has been in existence some time, a number of secondary cysts may form in it. The mode of origin of these is not in all cases quite certain; they are described both as arising from

vesicular transformation of the scolices, and as derived from the wall of the brood capsule. They are called daughter cysts. Each finally has an outer laminated, chitinous layer, an inner cellular layer, and a central collection of hydatid fluid. Each daughter cyst may in turn give rise to fresh scolices and to a fresh generation of daughter cysts; so that we may ultimately get almost innumerable cysts within cysts within the original parent cyst, and the process is further complicated by the fact that daughter cysts are sometimes formed from scolices which never became inverted, so that these daughter cysts are formed outside the cyst with which they are connected.

Hydatids nearly always grow in man in the manner just described; but commonly in the pig and rarely in man the growth is exogenous—that is to say, the original brood capsules grow as external projections on the outside of the parent cyst and not as internal projections into it. These new cysts outside the parent cyst soon become detached from it. A second generation of cysts forms from them; these become detached, and a third generation forms from them, and so on. Thus we get what has been termed the burrowing type of hydatid, in which cysts, forming in the direction of least resistance, form rows—often of great length—in between the muscles and fascia, so that the last-formed cyst is at some distance from the first. This exogenous form of

growth is not seen in children, and, as just remarked, is rare in the human subject; but I have seen it lead to the formation of innumerable cysts in the pleura, the original cyst being in the liver. It should be mentioned that very rarely a projection originally exogenous becomes inverted into the parent cyst, which may thus become packed with cysts that are really exogenous; and lastly, the same cyst may give rise to both exogenous and endogenous cysts. When exogenous growth takes place in man it is usually in the bones. In the viscera, *e.g.* liver or lungs, growth is nearly always endogenous. In a few cases neither scolices nor daughter cysts develop from the original cyst, which is then said to be sterile.

The cycle is completed by the dog eating offal from some animal, *e.g.* pig, sheep, oxen, containing hydatid cysts with scolices, which in the intestine of the dog grow into the *tænia echinococcus*, with which this description began.

This parasite also exists in the alimentary canal of the wolf, jackal, and dingo, the wild dog of Australia; but the food and drinking-water of man is hardly likely to be often infected with *faecal* matter from any host of this worm except the dog, and it is in Iceland and Australia, where dogs are brought into closest contact with man, that hydatid disease is commonest. Other animals besides the pig, sheep, and ox harbour the hydatid stage of development of

tænia echinococcus; they are man, monkeys, lemurs, deer, camels, giraffes, horses, asses, zebras, kangaroos, squirrels, seals, cats, turkeys, and peacocks. That this account of the life cycle of *tænia echinococcus* is, in the main, correct has been shown by feeding dogs on the flesh of sheep, oxen, and pigs containing hydatid cysts and by feeding them with hydatid cysts obtained from man, the *tænia echinococcus* developing in dogs so fed. On the other hand, the giving of the proglottides of *tænia echinococcus* from the dog to certain of the above animals liable to hydatids has caused hydatid disease in them.

In Iceland 1 person in 27 has hydatid disease, and in the Mount Gambier Hospital in South Australia 1 patient in every 65 has hydatid disease; in the northern parts of Australia it is almost unknown. During the last nineteen years there have been in Guy's Hospital 43 cases of hydatid of the liver and probably 4 or 5 hydatids of other organs. In St. Bartholomew's Hospital 1 patient in every 1100 admitted has hydatid disease.

I have seen hydatid of the liver in children, but the patients are usually adults. The ratio of the sexes will depend upon their relative likelihood of drinking contaminated water or eating contaminated food. In Australia hydatid disease is commoner in men; in Iceland, in women. Usually there is only one original cyst in the liver derived from a single embryo, although, of course, there may be numerous

brood capsules and daughter cysts derived from this one; but there may be two original cysts in the same liver, and in the last patient under my care there were probably three. It is important that at the time of operation the surgeon should, as far as possible, determine whether more than one cyst is present.

Symptoms.—Hydatid disease of the liver can hardly be recognised unless the cyst causes a discoverable tumour of the liver. This may be huge. The largest is recorded by Robinson; it contained 36 pints, occupied three-fourths of the abdomen, and was successfully removed. It had been previously tapped and 70 pints withdrawn. If the tumour can be felt, it is rounded and smooth. It may project from the under-surface of the liver, and thus suggest a dilated gall bladder, or, if it grow up from the upper surface of the liver, it presses up the diaphragm and may simulate a considerable pleuritic effusion. As the right lobe is larger than the left, hydatid cysts are commoner in the right than the left. If the cyst has not come to the surface, it may cause a rounded swelling composed of hepatic tissue; but under all circumstances the distinguishing features of hydatid disease of the liver are that the tumour is localised, regular, round, and smooth, and thus by its feel it can be told from the localised tumours formed by masses of growth or gummata, for these localised tumours are irregular, not so spherical, and often not smooth

The hydatid tumour is not tender, nor painful, and thus differs from an abscess, which also forms a regular, rounded and smooth localised tumour. If the hydatid tumour come below the ribs it may be discovered by palpation, and the features of it are much more easily distinguished if the patient takes a deep breath. If the cyst is large and causes considerable swelling of the right lobe in its lower part, the whole of the palpable part of the right lobe may be increased in size, and the hepatic dulness is prolonged downwards. If, on the other hand, there is a considerable hydatid cyst which grows upwards from the upper surface of the liver, a dome-shaped area of dulness is added to the upper limit of the hepatic dulness. As has just been mentioned, this may be so considerable that its dome-shaped character is lost, and then the chest may be considered to be full of fluid. A hydatid of the left lobe is said to have been mistaken for pericardial effusion, and a large hydatid of the right lobe may cause bulging of the lower part of the right chest, visible to the eye and demonstrable to measurement with a tape. In the case of great destruction of the right lobe by a large hydatid cyst, the left lobe may be enlarged from compensatory hypertrophy. If the hydatid is deep in the liver, and yet the swelling caused by it is palpable, this feels hard and solid; but if the abdominal walls are thin and the tumour has come to the surface, the

tumour feels tense and elastic. Occasionally fluctuation may be obtained; the cyst, however, is nearly always too tense for this. To obtain what is usually termed a hydatid thrill, one of the fingers of the left hand is placed over the tumour and percussed with the right forefinger; if the sign is present a thrill is then felt by the finger of the left hand. This sign is of little use, for it is very rarely obtained, and, further, it may be felt over any tense collection of fluid; but as hardly any collections of tense fluid, except hydatid cysts, occur in the liver when obtained over this organ, it is very considerable evidence in favour of hydatid. Naturally the diagnosis of hydatids is more difficult if more than one hydatid tumour is present in the same liver. It is excessively rare for hydatids to cause symptoms by pressure on neighbouring structures within the abdomen; for example, jaundice hardly ever results from pressure on the bile ducts. If jaundice is present it nearly always means that the hydatid cyst has ruptured into the biliary passages. If ascites is present, it is due to some complication, and not to pressure on the portal vein; symptoms due to pressure on the vena cava are excessively uncommon. But a large cyst growing upwards will cause symptoms of compression of the lung and may displace the heart.

Often there are no symptoms, and the patient may be quite unaware that he has anything the

matter with him until the tumour is accidentally discovered. In a few cases he complains of a sensation of weight and dragging. There is no pain, unless there is some local peritonitis over the cyst, and this is rare. In a few instances there is an increase of eosinophile cells in the blood. I have seen 10 per cent. present, and even the large number of 50 per cent. has been recorded; and I have known the percentage of eosinophile cells to drop by four-fifths within a few days of the cyst being opened. It is said that the degree of eosinophilia depends upon the activity of the hydatid. Although eosinophilia is often absent, still in a doubtful case of hepatic tumour its presence is a point in favour of hydatid; but it must not be forgotten that it may be present in cases of malignant disease. Reference has already been made to the fact that if any of the hydatid fluid becomes absorbed into the general circulation, the patient may have urticaria. Some authors, especially Welsh and Chapman, have pointed out that, if a sufficiency of blood serum from a patient with hydatid disease is mixed with hydatid fluid and allowed to stand at the temperature of the room for about twenty hours, a precipitate appears, but it does not if the hydatid fluid is mixed with blood serum from a patient who has not got hydatid disease. Not all hydatid fluids give the reaction; but if some hydatid fluid, which gives a precipitate when mixed with the

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blood serum of the patient from whom it was taken, is used, it will always give a precipitate when mixed with the blood serum of any patient infected with hydatids, but not when mixed with blood serum of those who are not so infected.

A few cases of hydatid of the liver are complicated by hydatid disease of other viscera. Thus out of 1000 patients dying in the Adelaide Hospital, South Australia, 49 had hydatids, and 11 of these had hydatids in more than one organ; in 5 two organs were affected, and in 6 three or more. In a very complete table compiled by Thomas and containing nearly 1900 cases of hydatid, in 57 per cent. the disease was in the liver, in 11·6 per cent. in the lungs, in 4·7 per cent. in the kidney, in 4·4 per cent. in the brain, in 2·1 per cent. in the spleen, in 1·8 per cent. in the heart, and in the peritoneum in 1·4 per cent.

The Natural Course of the Disease.—The connective tissue covering of the cyst may undergo degenerative changes. It becomes fibrous and tough, and in places it may occasionally be calcareous, due to the deposition of phosphate and carbonate of lime, and in very unusual cases degenerate bone tissue forms, and it has been known that the capsule could only be cut with a saw. Lardaceous, gummatous, and malignant deposits have all been known to occur in this capsule,

The outer laminated wall of the sac increases in thickness; fresh layers form, so that after some time the lamination is very evident; and when this wall is torn individual laminae may turn up at the edge of the tear. This lamination is so characteristic of hydatid cyst that the presence of a piece of laminated membrane, even so small as to be only well seen with a microscope, is conclusive proof of the presence of hydatid disease, and the presence of hooklets in any fluid shows it to be hydatid fluid. About a quarter of all the hydatid cysts found in the post-mortem room have undergone spontaneous death. The life of hydatid cysts may, however, be considerable. A man was for twenty-two years thought to have a sebaceous cyst of the skin, but it was found to be a live hydatid.

The cause of aseptic death of hydatid cysts is not well understood, but sometimes, at least, it appears to be due to the escape of bile into the cyst. Aseptic dead hydatid cysts always contain bile or some derivative of it. The first stage in the death and spontaneous retrogression of a hydatid cyst is shown by the fact that the hydatid fluid of the original cyst becomes turbid owing to the presence of insoluble proteids. The fluid of the brood capsules and daughter cysts is not affected till later; but by the time it becomes turbid that of the original cyst has come to contain a quantity of smegma-like fatty material; the walls of the original cysts become

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gelatinous; they shrink and are thrown into folds. Later the contents and walls of the brood capsules and daughter cysts undergo the same change. As the degeneration progresses we find the contents of the original cyst, brood capsules and daughter cysts, converted into a whitish smegma or putty-like fatty mass, imbedded in which are the gelatinous degenerate capsules of brood cysts and daughter cysts. The fatty material contains stearin and cholesterin crystals and various other crystals. In the last stage, which is rarely reached, the fatty mass becomes calcareous, but its hydatid origin is revealed by the fact that hooklets may be found in it. The degenerate contents of hepatic hydatid cysts are often stained green from the presence of biliverdin; traces of bilirubin may be present also in the form of crystals.

Small papillomatous excrescences may be seen on the inner surface of a few cysts, and these, like the rest of the wall of the cyst, may undergo gelatinous degeneration. These excrescences are derived from the cellular layer, and represent abortive attempts at the formation of brood capsules. Lastly, in some quite exceptional cases the wall of the original cyst disappears; how it is absorbed we do not know.

Rupture.—Hydatids of the liver which do not die may rupture, but happily it is not common for them to do so. Rupture may take place into (a) the peritoneal cavity, and is then usually due to a blow

or other form of injury. If the hydatid is suppurating, rupture into the peritoneal cavity is almost certainly quickly fatal; but if it is not suppurating the result is variable, depending, no doubt, upon whether the hydatid fluid is very poisonous. If the fluid is very slightly poisonous and the amount which escapes is small, there may be no symptoms, or the symptoms may be only a little urticaria; on the other hand, if the fluid is abundant and toxic, there may be profound cardiac depression and fatal collapse. If the patient was not known to suffer from hydatid disease, the urticaria and collapse might well give rise to a suspicion of irritant poisoning. If the fluid is abundant and the patient survives, the signs of ascites may be detected; or again, if he survives, the peritoneum, especially that of the omentum or pelvis, may become infected by the daughter cysts, and so subsequently he may suffer from hydatid disease of the peritoneum. Lastly, several small granulomata may form on the peritoneum; each of these contains some hooklets or pieces of membrane, and the granulomata no doubt result from the irritation of these foreign bodies. This condition has been called pseudo-tuberculosis of the peritoneum. Occasionally a hepatic hydatid cyst already communicating with the bile passages ruptures into the peritoneum, which then contains bile-stained fluid.

Rupture may also take place into (b) the lung when

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adhesions have formed between the hydatid and the diaphragm, and between the diaphragm and the lung. This is a serious complication and may lead to the death of the patient from suffocation. Hydatid cysts may be expectorated, and if the hydatid contains bile the expectoration may be bile-stained. Pneumonia and gangrene of the lung are both liable to follow.

Rupture may occur into (c) the pleura. This is not so serious as the last, especially if it is recognised before rupture into the lung occurs. The physical signs are those of fluid in the chest. This is readily known not to be ordinary pleuritic fluid by the fact that it does not coagulate on heat. I have known failure to recognise that some fluid removed from the chest by aspiration was hydatid fluid lead to most serious results, and it should be made a matter of routine always to see if such fluid coagulates on heat; if it does not, microscopical examination of the centrifugalised deposit will probably reveal the presence of hooklets and little pieces of laminated membrane. Whether an empyæma follows rupture into the pleura depends upon whether the hydatid was suppurating. If rupture takes place into the lung, a pneumo-thorax or pyo-pneumo-thorax may form, and very rarely the fluid in the chest contains bile.

Rupture may also take place (d) into the pericardium. This is merely a pathological curiosity, and is always quickly fatal. (e) Into the bile ducts.

A few cases are known in which this has occurred, and the passage of the cysts down the common duct has led to biliary colic, the true cause of which has been determined during life because the cysts or pieces of hydatid membrane have been found in the fæces; but unless these have been found, the case is almost certainly regarded as one of biliary colic due to gall-stones, for the pain and jaundice due to this form of obstruction of the common duct are exactly the same as when caused by gall-stones. Cholangitis is likely to result, and, if the hydatid fluid is sterile, this is due to infection from the duodenum.

Rupture into (*f*) the stomach or intestines. This is not very rare. If it takes place into the stomach, hooklets, pieces of membrane, or cysts may be detected in the vomit; if into the intestines, in the fæces. A patient under my care was operated on for a hydatid of the left lobe of the liver. He died from suppuration of another hydatid cyst in the right lobe. At the post-mortem examination it was found that the cyst in the left lobe had, some time previous to operation, communicated by a narrow opening with the duodenum. The discharge of a hydatid into the gastro-intestinal tract is often followed by complete recovery. It may lead to the presence of gas in the hydatid cyst. (*g*) Rupture into the inferior vena cava or hepatic veins has occurred, and is sometimes almost immediately fatal, for the cysts being carried to the lungs cause pulmonary

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embolism, but rupture into veins is very rare. (*h*) Externally. This is the most favourable place for rupture, but it can only occur when the cyst is superficial, and it will then most likely be detected and operated upon before rupture occurs.

Suppuration.—Hydatid cysts of the liver not infrequently suppurate. It has been suggested that this may be caused by the irritating character of hydatid fluid, but it is probably always microbic. It may follow puncture of the cyst, or infection of the cyst after rupture into the bile passages or gastro-intestinal tract, or infection may take place as a result of the patient having some specific fever, *e.g.* typhoid; but in most cases it is not clear why the cyst should suppurate. When suppuration occurs, the case becomes one of abscess of the liver, and the symptoms are the same as have been already described when describing solitary abscess of the liver; and we have, when speaking of subdiaphragmatic abscess of the liver, pointed out that such an abscess may be a suppurating hydatid, and that then it usually is present at the part of the liver that is uncovered by the peritoneum. If more than one hydatid cyst is present in the liver, it may be that one suppurates although the other does not.

Complications.—Hydatids may be present in other organs, but the symptoms due to this hardly fall within the description of hydatid disease of the liver. Other complications are produced by rupture

of the cyst in various directions and by its suppuration, but these have been just described.

Diagnosis.—Hydatid tumours of the liver are distinguished from other tumours of the liver by the fact that they are, unless suppurating, neither painful nor tender. The tumour formed by them is rounded and smooth; evidence of fluid in it may occasionally be obtained by physical examination. The swelling of the liver is local, and the liver is not uniformly enlarged. The most common tumour of the liver is that produced by malignant disease, and there are many points, such as wasting, jaundice, evidence of the primary seat, the irregular hard feel of the tumour, which will usually lead us aright. A suppurating hydatid can hardly be distinguished from a solitary abscess of the liver except by the history of the case. Syphilis of the liver usually causes the organ to be much deformed, and the swelling of it is very irregular and not smooth. When a simple cyst of the liver is large enough to be detected during life, diagnosis from a hydatid is very difficult (see p. 284). A hydatid projecting from under the lower edge of the liver may closely resemble a distended gall bladder. A swelling in the abdominal wall may usually be distinguished from a hydatid of the liver by physical examination; it does not move up and down on respiration, as does a hydatid of the liver. Confusion has occurred between hydatid of the liver

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and pleural effusion, but the dulness is often dome-shaped when due to a hydatid of the liver, which is more likely to cause bulging of the chest than pleural effusion, for this so often causes considerable collapse of the lung. Pleural effusion is much more likely to displace the heart, and it more easily leads to impaired movement on the right side. The fluid of pleural effusion coagulates on heating; that from a hydatid cyst does not. Sometimes hydatid is associated with pleural effusion, and hydatids of the liver which have by growing exogenously entered the pleural cavity have been mistaken for pleuritic effusion; but the absence of albumen in the fluid withdrawn will show the hydatid nature of the fluid. Floating kidney, hydro-nephrosis, and pancreatic cyst can all usually be distinguished if sufficient care is taken, and mistakes are less likely to be made if the possibility of them is always borne in mind. Examination with the X-rays will often help.

Treatment.—Many methods of treatment have been adopted, but only two need be mentioned. Until a few years ago the cyst was usually punctured by a fine aspirating needle and some fluid drawn off. This often cured the patient by leading to the death of the hydatid, but for the following reasons it should not be employed. It sometimes does not lead to the death of the hydatid, and it is impossible to tell beforehand whether it will. Even if it does, the

dead hydatid is left behind and may at any time suppurate. The puncture of the cyst has led to suppuration in it. Symptoms of serious and even fatal collapse have often followed puncture, probably the explanation of them is that some of the hydatid fluid escapes, through the hole made by the needle, into the peritoneum, pleura, or lung, according to the situation of the puncture, and we have seen that some specimens of hydatid fluid are powerful poisons, causing urticaria and even cardiac collapse. Important structures may be damaged by puncture; thus the peritoneum, pleura, lung, portal vein have all been seriously hurt.

It is generally acknowledged that the only right treatment is to expose the cyst and deal with it in the best way that the particular case suggests. Some advocate stitching it to the skin and letting the cavity granulate up after the cyst is removed; others prefer to remove the cyst, sew up the hole in the liver left by it, and drop the liver back into the abdomen, but for the details the reader must consult books on surgery.

Hydatid disease is rare in England, and therefore little trouble is taken to prevent it. But it may be brought about even here by too close contact with animals, as in the case of a lady who repeatedly became infected owing to the disgusting habit of constantly fondling and kissing a pet dog.

Human beings are nearly always infected by

drinking-water; therefore in countries in which hydatid disease is common all drinking-water should be boiled or efficiently filtered, and all raw vegetables should be scrupulously washed with boiled water before being eaten. Further, as infection almost always takes place from dogs, the number of these should be kept under by the destruction of all dogs for whom no licence has been obtained, and dogs should not be fed upon such offal as is likely to contain hydatids, or, if they are given meat, it should be cooked, for hydatids are killed by cooking the meat which they infect.

ALVEOLAR ECHNOCOCCUS DISEASE

This is sometimes known as multilocular echnococcus disease. It is very rare—no case has, as far as is known, occurred in England—and very dangerous to life. Of all the organs in the body the liver is by far the most frequently affected. That the disease is parasitic there is no doubt. Virchow considered that it was due to the same parasite as ordinary hydatid disease, but that it grew in an unusual way. Most authorities do not now hold this view, which is very hard to understand for many reasons, for example, alveolar echnococcus disease does not occur in the same countries as ordinary hydatid disease; probably alveolar echnococcus disease is due to a different species of parasite.

The following description is abstracted from that by Stirling and Verco. Occurring both in man and some animals with by far the greatest frequency in the liver, where the tumour may attain any size up to that of an adult's head, it nearly always leads to a fatal result. In its broad structural features the main mass of such a tumour consists of a vacuolated or sponge-like stroma of sclerosed connective tissue more or less infiltrated with lime salts. This disposition of its framework defines numerous alveolar cavities, varying in diameter from 1 to 5 mm., which are filled with plugs of colloid material, consisting of plicated and crumpled chitinous vesicles, having the same laminated structure and chemical composition as those of ordinary hydatid cysts. Both surfaces of the individual folds of the vesicles are covered with a finely granular layer of protoplasm associated with other parasitic elements, such as fully-formed scolices or detached hooklets. Such a primary tumour may give rise to neighbouring and satellite growths in the same viscus, or to metastases in distant organs. Before it attains any considerable size, which only happens in the liver, retrogressive and necrotic changes, aided by septic infection and bile irruption, produce towards the centre of the growth a ragged and anfractuous cavity filled with bile-stained, sero-purulent fluid, holding in suspension calcareous particles, cholesterin, bilirubin, and other detritus.

The biological relationships of this tumour are so little understood that a discussion about them would be out of place here, but the most recent view is that the contorted chitinous vesicle is the homologue, not of any ordinary echinococcus bladder, but of a ripe segment of a *tænia*, because, like the latter, it can produce living embryos; these again are capable of repeating the activities of the parent, either in adjacent tissues or distant organs. Hence the malignant characters, and at the same time the explanation of the multilocular character of the tumour.

The intermediate host is not known. Out of 235 cases 214 occurred in Bavaria, the Tyrol, Switzerland, Würtemberg, and Russia. No cases have been recorded from Iceland or Australia.

Jaundice is very common. Sometimes there are no gastro-intestinal symptoms; at other times they are severe. The disease is slow, the jaundice deepens and may last two years. The usual symptoms of chronic jaundice are present. The liver is enlarged. The patient gets weaker and gradually sinks, with pyrexia if suppuration of the cyst is present. Hitherto treatment has been very unsatisfactory, for the disease is not usually diagnosed until the tumour is so large that it has been impossible to attempt its removal, or if the removal has been attempted the operation has been fatal.

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OTHER CYSTS OF THE LIVER

Cysts of the liver not hydatid are very exceptional. Moschcowitz, writing in 1906, could only find eighty-three cases in medical literature. There are two varieties. First, those not associated with cysts elsewhere. These are of all sizes, and in rare instances have contained many pints. The liver may contain any number, from one to dozens. Usually when multiple they are numerous, the organ on section looks like a honeycomb, and may have on its surface the projections due to the numerous cysts. In an extreme case the liver may weigh several times its normal weight; one weighed 35 lbs. Probably these cysts are due to obstruction of small bile ducts, and after a time the colouring matter is absorbed and only clear fluid remains; but, generally speaking, the obstruction cannot be demonstrated. Sometimes hæmorrhage takes place into them. They are surrounded by a firm fibrous capsule, and the lining membrane is smooth. The walls between contiguous cysts may break down, and thus give a reticulated appearance. Until the fluid from these cysts is examined, it cannot be distinguished from hydatid fluid.

A special variety of these cysts is the solitary non-parasitic cyst. This is very rare, may attain great size, and has a thin strand of hepatic tissue over it. Mr. Bland-Sutton suggests, and probably correctly, that these solitary cysts arise from dilatations of the bile ducts which subsequently fuse to form a single cyst. They are only found in women and at the free edge of the liver, and are perhaps associated with tight-lacing. He records the case of a woman from whom such a cyst, containing two pints of fluid, was removed. It is clear that the difficulties of diagnosis must be very great.

The second variety of hepatic cysts are usually spoken of as "Cystic disease of the liver," and are almost always associated with cystic disease of the kidneys, and in 19 per cent. of all cases of cystic disease of the kidneys cysts are also found in the liver. These cysts are always congenital, even when found in adult life. It is rarely that they are suspected during life, but when found in infants they are often associated with various deformities. They are commoner in females than in males. Usually they are excessively numerous, easily visible to the naked eye, form rounded swellings on the surface of the liver, and lead to a great increase in its size and weight, but in a few cases they are only found on microscopical examination. They contain clear fluid. This disease of the liver gives rise to no symptoms unless its

size is great. The urine is pale, of low specific gravity, and nearly always contains albumen. The mode of origin of these cysts is very obscure, but it is believed that they are associated with aberrant bile ducts. Occasionally other organs than the kidneys and liver are affected. Dr. Savage and I have come across two general paralytics in whom cysts were found in the liver, kidneys, lungs, heart, and brain.

Foaming Liver.—After death the liver is occasionally found to contain a large number of small cavities or cysts which are full of gas. When this occurs the organ is a little lighter in colour than natural, it is soft, and when handled gives rise to an emphysema-like crackling. This state of the liver is usually due to the formation of bubbles of gas in it by the *bacillus aërogenes capsulatus*, but Pakes and Bryant have recorded a case in which the production of a foaming liver was due solely to the *bacillus coli communis*. In some cases both these organisms have been present, and some have been recorded in which the *bacillus maligni oedematis* was found. Under the microscope these various bacilli can be seen in large numbers round the bubble, and they often produce bubbles of gas in other organs besides the liver; but although the liver is not necessarily affected when other organs are, it is most commonly affected. Among 22 cases collected by Pakes and Bryant, in which

these cysts were present in some part of the body, in 2 they were universal, the liver was affected in 15, the spleen and kidney each 9 times, the myocardium in 6, the stomach and intestine in 3, the penis and scrotum, lung and heart each 2, and the uterus, supra-renals, urinary bladder, skin of neck, chest and axilla, serous cavities and coronary arteries once each. Usually the formation of these gas-containing cavities begins during the agony, or more probably shortly after death, and continues until the post-mortem examination is made; thus they are more often found if some considerable time has elapsed between death and the post-mortem examination, and consequently I have noticed that a foaming liver is more often found in the post-mortem room on Monday than any other day in the week, and it is more common in hot weather. The bacillus *aërogenes capsulatus* is anaërobic, and is widely distributed in nature. Pakes and Bryant were of opinion that in their case, due to the bacillus coli, the formation of these gas-containing cavities had commenced before death.

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ACUTE YELLOW ATROPHY

THIS extremely dangerous disease is fortunately seldom seen. In twenty-five years only seven cases occurred at St. Bartholomew's Hospital, and in twenty-seven years there were eleven cases at Guy's Hospital. It is difficult to resist the belief that the cause must be a virulent micro-organism, and the appearance of the liver is so similar in all the fatal cases as to suggest that the micro-organism is the same in all cases, but it is only fair to add that some authors have suggested that different micro-organisms may be the cause in different cases. In some way that is not understood pregnancy predisposes to the disease; indeed, so many of the cases occur during pregnancy—usually between the fourth and seventh month—that the proportion of women to men who have the disease is about 2 to 1. The occurrence during pregnancy also influences the age incidence, for half the cases occur between 20 and 30, and four-fifths of the cases between the ages of 10 and 40. A few cases have been published as occurring in young children, but it is doubtful whether all of them were examples of

acute yellow atrophy. The disease is so fatal and so strange in its symptoms, post-mortem appearances, and association with pregnancy, that, although so rare, it is well known ; and hence, when a pregnant woman becomes jaundiced the doctor becomes very apprehensive, and I have seen two or three cases of jaundice due to other causes, *e.g.* gallstones or catarrh, and associated with pregnancy, and in which for a few days there has been great alarm as to whether the case was one of acute yellow atrophy. Sometimes the disease appears to be directly traceable to syphilis, but as acute yellow atrophy itself is so exceptional and only some cases are the sequel of syphilis, post-syphilitic acute yellow atrophy is very rare. It is important to bear this in mind, and not to jump to the conclusion that a patient is going to have acute yellow atrophy just because he happens to be jaundiced when he has syphilis, for a harmless jaundice, which quickly yields to treatment, is a well-known accompaniment of secondary syphilis. In a few cases over-indulgence in alcohol and pre-existing hepatic disease have been antecedents of acute yellow atrophy, and it is only likely that in such a case the resisting power of the liver to micro-organism of acute yellow atrophy is especially low. Much confusion has arisen because some have suggested that acute yellow atrophy is really due to phosphorus poisoning, but this suggestion shows

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a lack of experience at the bedside and in the post-mortem room. In no case of acute yellow atrophy that I have seen has there been any suggestion of phosphorus poisoning, and although towards the end of the case the symptoms are somewhat similar, yet in the early stages the gastrointestinal symptoms are much more marked in phosphorus poisoning. Then, too, in phosphorus poisoning the liver is almost always enlarged, and at the post-mortem it is of a pale yellow, with a very great increase of fat; in acute yellow atrophy the liver is small, and of a bright yellow with splashes of red, and it is not particularly fatty.

The sufferer from acute yellow atrophy usually first feels ill without any definite symptoms, and then in two or three days she becomes jaundiced; in this early stage the condition resembles a severe attack of catarrhal jaundice, but the patient is, as a rule, more ill, and there is some rise of temperature, but usually by the end of a week, or at the outset ten days, the diagnosis becomes clear, for instead of mending, as a sufferer from catarrhal jaundice would do, the patient sinks into a typhoid condition. She becomes obviously very ill, the jaundice persists and perhaps deepens, the pulse is rapid and small, the temperature is raised but irregular, for it often drops to below normal. Head-ache is now severe, vomiting is very distressing, and often the vomit contains blood; there is great

restlessness with delirium, muscular twitchings are common, and there may even be convulsions; the pupils are dilated, the mouth is dry, there are sordes on the lips and tongue, which is very dry and furred, hæmorrhages are seen on the skin, in the mouth, and on the lips, respirations are a little quickened. Hour by hour the symptoms become more grave, the pulse becomes more rapid and feeble, the mouth is very dry, blood collects on the teeth and lips, the patient becomes drowsy and apathetic, sinks in the bed, and passes her motions and urine under her. Subsultus is often seen; more hæmorrhages appear on the skin, more blood in the vomit, and the motions, which smell foully, contain blood. The patient aborts, becomes more and more apathetic, and finally comatose; the temperature is now often subnormal, and she finally sinks and dies, often within three or four days after the typhoid symptoms supervene. There is perhaps some rise of temperature immediately before death. The first stage usually lasts about a week, and the typhoid stage three or four days.

During the typhoid stage percussion shows a rapid diminution of the size of the liver, the jaundice deepens, the urine, which has contained some bile from the first, becomes darker, for the amount of bile in it increases. The quantity of urine passed is diminished, albumen may be present in it, but no

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sugar is found. The most striking characteristics are that the amount of urea excreted is greatly diminished, but the quantity of ammonia is increased; leucin and tyrosin can often be found in the urine, and if not at first discoverable, they may be discovered after the urine has been concentrated by evaporation. Leucin appears in the form of discs, and the crystals of tyrosin are needle-shaped.

The above account represents the symptoms met with in the great majority of cases, but in very unusual instances the disease is prolonged for some weeks; in others the liver does not atrophy much. It may be that no leucin or tyrosin can be found in the urine, and in exceptional cases jaundice does not occur.

The prognosis is very grave; a few cases have been said to recover, but it is probable that some of them were not examples of acute yellow atrophy. As far as we know there is no treatment that will cure or retard the disease.

When a post-mortem examination is made on a case of acute yellow atrophy the liver is seen to be extraordinarily shrunk. It may weigh less than half its usual weight. The shrinkage is uniform, but the small size of the left lobe is often very striking. The atrophy of the liver leads to wrinkling of the capsule, which can be picked up with a pair of forceps off the liver, and, as might be expected, considering the rapidity of the process—for

the liver may lose half its weight in ten days—the organ is flabby and has lost its natural firmness, so that when put on the table it lies more or less in a heap. This flabbiness of the liver is partly responsible for the diminution of hepatic dulness, for because of it the liver falls away from the abdominal wall. Parts of the liver are usually of its natural colour, but rather darker; parts are dark greenish-yellow—the two merging one into another. Here and there splashes of red are seen and also minute hæmorrhages under the capsule.

When the liver is cut the same kind of resistance is felt as is experienced in cutting a collapsed lung—the organ is not soft; indeed, it is rather tough when pressed by the finger. The appearance of a section is one of the most striking objects seen in the post-mortem room. The prevailing colour is bright yellow, but it is interspersed with patches of red. The degenerative process is further advanced in the red patches than in the yellow; hence in the red areas the outlines of the nodules cannot be discerned and the red areas are more numerous the longer the case has lasted; much of the necrotic debris of the hepatic cells has become absorbed from these red areas, and the numerous remaining capillaries give the red colour, and the red areas may be a little depressed below the yellow. Early in the course of the disease the lining membrane of the minute bile ducts swells—hence the jaundice—

and consequently in the post-mortem room the larger ducts contain nothing but mucus. The total amount of fat in the liver is a little increased, but not nearly enough to give either the appearance or feel of a fatty liver. A scraping of the surface of a section of the liver shows many degenerate cells and crystals of leucin and tyrosin. Some authors have described that when the illness has been prolonged for many weeks—which is excessively seldom—adenomatous nodules resembling new growths may be seen; they should be regarded as a compensatory growth of new hepatic cells; indeed, they are exactly similar to the multiple adenomata met with in cirrhosis.

The histological appearances are those of very rapid degeneration. Some of the hepatic cells are fatty, others are granular and bile-stained, others show degeneration of their nuclei; minute hæmorrhages are numerous; the walls of the smallest bile ducts are inflamed. In the red areas the process is advanced still further, for the broken-down debris of the cells has been absorbed and only the framework of the lobule remains, and, indeed, it may be difficult to tell that the section is one of liver at all. Here and there some attempt at repair may be seen.

The organs of the rest of the body show the changes usually met with in an acute infective disease; thus the kidneys are swollen, the spleen is

soft and a little enlarged, the cardiac muscles show cloudy swelling; minute hæmorrhage may be seen in many organs, and the viscera are bile-stained as in any other case of jaundice. As far as we know, no treatment is of any avail.

Icterus Neonatorum

This phrase is used to describe jaundice which occurs in infants at or shortly after birth. Clinically there are two varieties—that which is harmless and soon passes off, and that which indicates dangerous disease.

The harmless variety, often called yellow gum, is very common; it is said that about a half or a third of all babies suffer from it. The condition is more frequent among the poor than the well-to-do. The jaundice comes on within the first five days of life, begins to fade in two or three days, and completely disappears in a week or two. Curiously the sclerotics are said to be not affected till after the skin. In the commoner mild cases the urine often does not contain bile, and bile is usually present in the fæces. The staining of the skin is like that of any other case of jaundice. The gums are dyed yellow, hence the popular name.

If the sufferer from it accidentally dies from some injury or illness distinct from the jaundice, all the organs in the body are stained yellow except the liver, spleen, and kidneys. Even the nervous system may be so stained. No treatment is

required. The cause of the trouble is believed to be an excessive viscosity of the bile.

Dangerous jaundice in the new-born may have several causes. It may be due to portal pyæmia, which arises by infection of the umbilicus, and then there is suppurative phlebitis of the umbilical vein. The infection is usually streptococcal, and the symptoms are those of portal pyæmia (see p. 52); but as the patients are children, rigors do not occur. The disease is nearly always fatal. Fortunately it is much less frequent than it was. This is because the importance of asepsis and cleanliness is now properly understood. Infants, like adults, may suffer from general septicæmia accompanied by jaundice, and it has been recorded that epidemics of septicæmic jaundice have occurred in the new-born. To this variety of jaundice the name Winckel's disease has been given, because he described such an epidemic; happily they are very rare.

Congenital obliteration of the bile ducts is another cause of serious jaundice in the new-born. Jaundice is present at birth or shows itself within the first few days of life. It slowly becomes very dark, the urine contains much bile, and the fæces are white and offensive. The liver is much enlarged, purpura is common, indigestion is severe, ascites and oedema develop; the baby dies from asthenia or convulsions usually when six or eight weeks old. The liver is found to be extensively cirrhotic, the

cirrhosis is chiefly monolobular. There is also considerable cholangitis with obliteration of the minute bile ducts. The cause of the trouble is unknown; there are several instances in which most of the children of the same parents have been afflicted.

Lastly, infants are sometimes born with a congenital obliteration of the bile duct at its entrance to the duodenum, or with gall-stones, or with syphilitic disease of the liver, so severe as to cause jaundice within a few days of birth; but such occurrences are so exceptional as to be among the curiosities of medicine.

Weil's Disease

This is a specific fever of which jaundice, enlargement of the liver, and enlargement of the spleen are characteristic symptoms. It is hardly ever seen in Great Britain, and is met with chiefly in Germany and Russia. The onset, which is sudden, is very like that of influenza. There is a rise of temperature, accompanied by severe pains, especially in the calves of the legs and the head, and the patient feels ill. On the second or third day jaundice shows itself; it is generally slight, but does not finally disappear for about a fortnight. The temperature remains high, ranging about 103° or 104° , but with wide fluctuations, for a week, and then gradually it falls and the patient recovers. The liver and

spleen are both enlarged; the pulse is rapid, the motions are pale, and there may be albumen in the urine, which contains also bile, casts, and sometimes blood.

The disease is commonest in the summer months; it is believed to be due to the *bacillus proteus fluorescens*, and is perhaps in some way dependent upon decomposing meat, for several of the patients have been butchers, and it has been ascribed to eating sausages which have begun to decompose and have not been properly cooked. The sufferers nearly always recover, and no special treatment is needed.

Sometimes severe catarrhal jaundice, accompanied with some pyrexia, occurs in epidemics, when it is called infectious jaundice, and then confusion of diagnosis is likely to occur between it and Weil's disease. Yellow fever and relapsing fever, both specific fevers accompanied with jaundice, are so very rarely seen in Great Britain that they hardly call for description here. They are usually described among the specific fevers.

Emotional Jaundice

Many writers mention that persons may, under the influence of great fright or some strong emotion, become jaundiced, but such an event is most exceptional. Personally I have never seen a case, but I have been told of a case in which a girl became suddenly jaundiced on seeing her sister

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drown. This variety of jaundice has been attributed to spasm of common bile duct; it usually soon passes off.

Functional Disorders of the Liver

It is quite common to meet with persons said to suffer from "a torpid liver" or from "an attack of liver," or a well-educated man will be content with saying he knows what is the matter with himself, and he tells his doctor "he has a liver." The symptoms of this condition are a feeling of distension, or of a weight in the upper part of the abdomen, together with flatulence, constipation, and the usual symptoms of dyspepsia. Depression of spirits and headache are common. There is no evidence that these symptoms are due to disease of the liver; probably they are caused by some gastro-intestinal catarrh, the result of over-eating, and if a slight tint of jaundice is present, this may well be owing to a mild catarrh of the bile ducts caused by the spread of duodenal catarrh. The motions are often a little pale, but it is rash to assume from this that the liver is primarily at fault; sometimes this colour of the fæces is due to bubbles of gas, resulting from excessive carbo-hydrate fermentation. The condition is readily cured by plain simple diet, and by some simple aperient such as calomel. The fuller consideration of it belongs to disease of the gastro-intestinal tract.

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